TOBACCO AND HEALTH :

AN INDIAN PRESPECTIVE

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TOBACCO & HEALTH : An Indian perspective

Concern for tabacco-related diseases has been a major factor behind the prolonged stagnancy and often, decline in demand for tobacco products over the decade of the 1980s.

If the trend continues and control measures be intensified, a significant section of the Indian economy will become extinct.

With this background, in the following pages we have tried to explore various medical researches, patterns of Indian tobacco consumption and morbidity and mortality due to tobacco related diseases. The role of WHO and Indian control measures are also presented. Discussion sums up the findings and issues and makes recommendations.

International medical research

The suggestion of a statistical association between tobacco and cancer goes back over sixty years. Lombard and Doering (1928) in the United States, and Muller (1939) and Schairer and Schoeniger (1943) in Germany noted that smokers constituted a higher percentage of lung cancer patients than they did of controls. In 1950 the retrospective studies on smoking habits of lung cancer patients and controls by Schrek et al. (1950), Mills and Porter (1950), Levin et al. (1950), and Wynder and Graham (1950) produced evidence of the consistent association between smoking and cancer of the lung, a conclusion more firmly established by the better controlled studies of Doll and Hill (1952) in the United Kingdom and Hammond and Horn (1958) in the United States. Doll and Peto (1976) reported on general mortality among smokers as compared with controls. U.S. Surgeon General's reports and the reports of the Royal College of Physicians (1971, 1983) have summarised an enormous amount of material collected subsequent to these early studies.

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In the late 1950s the U.S. Public Health Service, The National Cancer Institute, The National Heart Institute, the Americam Cancer Society and the American Heart Association appointed a study group to examine the available evidence on smoking and health which led to the 1964 U.S. Surgeon General's report on Tobacco. This was a landmark which reviewed the available epidemiologic, clinical, pathological and experimental literature for evidence, linking cigarette smoking to disease. Cigarette smoking was found to increase the overall mortality, cause lung and laryngeal cancer, chronic bronchitis in men and suspected to cause cancer and increase the risk of pulmonary emphysema in women. The report of 1967 concluded that cigarette smoking would cause death from coronary heart disease and thus cigarette smoking was the most important cause of chronic non-neoplastic bronchopulmonary disease in the United States.

The 1968 and 1969 reports strengthened the conclusions reached in 1967. The 1971 report provided more detailed evidence on health consequences of smoking. Subsequent reports from 1972-76 revealed increasing association of cigarette smoking with many health hazards, with the 1973 report giving data on pipe and cigar.

The 1975 report updated knowledge on passive smoking. The combined 1977-78 report discussed smoking-related problems unique to women. The 1979 report discussed pipe, cigar and smokeless tobacco and the behavioural, pharmacologic and social factors influencing the initiation, maintenance and cessation of cigarette smoking. It found association of smokeless tobacco with oral cancer. Since the 1979 report, subsequent reports have focussed on specific (a) populations (women in 1980, work place in 1985), (b) diseases (cancer in 1982, cardiovascular diseases in 1983, chronic obstructive lung diseases in 1984) and (c) topics (low tar, low nicotine cigarette in 1981, involuntary smoking in 1986).

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The 1986 report of the Advisory Committee to the Surgeon General dealt with the health consequence of the use of smokeless tobacco and concluded that smokeless tobacco could be addictive and casue of oral cancer.

In 1987, the revised edition dealt with nicotine withdrawl

The 1988 report explored in detail the specific topic nicotine addiction, this being the drug in tobacco causing addiction. Moreover, the processes that determine tobacco addiction were similar to those that determine addiction to drug such as heroin and cocaine. It reported that nicotine was a powerful pharmacologic agent that acted in a variety of ways at different sites of the body. After reaching blood stream, it enters the brain, interacts with specific reactors in brain tissue and initiates metabolic and electrical activity in the brain. In addition, nicotine caused skeletal muscle relaxation and cardiovascular and endoctrine effects.

Like other addictions tobacco use could be effectively be treated.

The 1989 report concluded that cigarette smoking was a cause of cerebro-vascular disease and was associated with cancer of the uterine cervix.

A summary of the principal effects of cigarette smoking given in various Surgeon General's report and current claimed knowledge in 1989 is given below :

Figure - 1

Effect first discussed in Surgeon General's Reports	Year first discussed in a Surgeon General's Report	Current knowledge in 1989 (claimed)
Mortality and Morbidity		
Overall mortality, increased in men	1964	Overall mortality increased in men and women
Overall morbidity, increased	1967	Overall morbidity increased

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Figure - 1 (Contd.)

Effect first discussed in Surgeon General's Reports	Year first discussed in a Surgeon General's Report	Current knowledge in 1989 (claimed)
Cardiovascular		
CHD, mortality increased in men	1964	A major cause of coronary heart disease in men & women
Cerebrovascular disease (stroke), mortality increased	1964	A cause of cerebrovascular disease (stroke)
Atherosclerotic aortic aneurysm, mortality increased	1967	Increased mortality from atherosclerotic aortic aneurysm
Atherosclerotic peripheral vascular disease, risk factor	1971	A cause and most important risk factor for atherosclerotic peripheral vascular disease
Cancer		
Lung cancer, the major cause in men	1964	The major cause of lung cancer in men and women
Laryngeal cancer, a cause in men	1964	The major cause of laryngeal cancer in men and women
Oral cancer (lip), a cause (pipe smoking)	1964	A major cause of cancer of the oral cavity (lip, tongue, mouth, pharynx)
Esophageal cancer, associated with	1964	A major cause of esophageal cancer
Bladder cancer, associated with	1964	A contributory factor for bladder cancer
Pancreatic cancer, increased mortality	1967	A contributory factor for pancreatic cancer
Renal cancer, increased mortality	1968	A contributory factor for renal cancer
Gastric cancer, associated with	1982	An association with gastric cancer
Cervical cancer, possible association with	1982	An association with cervical cancer
Pulmonary		
Chronic brochitis, the major cause	1964	The major cause of chronic bronchitis
Emphysema, increased mortality	1964	The major cause of emphysema

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Figure - 1 (Contd.)

Effect first discussed in Surgeon General's Reports	Year first discussed in a Surgeon General's Report	Current knowledge in 1989 (claimed)
Women		
Low birthweight babies, associated with	1964	A cause of intrauterine growth retardation
Unsuccessful pregnancy, associated with	1980	A probable cause of unsuccessful pregnancies
Other effects		
Tobacco habit, related to psychological and social drives	1964	Cigarette smoking and other forms of tobacco use are addicting
Involuntary smoking, irritant effect	1972	A cause of disease, including lung cancer, in healthy non-smokers
Peptic ulcer disease, associated with	1964	A probable cause of peptic ulcer disease
Occupational interactions, adverse	1971	Adverse occupational interactions that increase the risk of cancer
Alcohol interactions, adverse	1971	Adverse interactions with alcohol that increases the risk of cancer
Drug interactions, adverse	1979	Adverse drug interactions
Nonmalignant oral disease, associated with	1969	An association with non-malignant oral disease
Smokeless tobacco, associated with oral cancer*	1979	Smokeless tobacco is a cause of oral cancer

* Smokeless tobacco includes all other types of tobacco consumed in ways other than smoking, e.g. chewing, snuff etc.

In 1962 the Royal College of Physicians, in a study came to conclusion that cigarette smoking caused premature deaths and disabling illnesses.

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Figure - 2

The following have been some of the other major studies on relationship between smoking and cancer in humans:

Study	Year of enrollment	Sample size: initial samples; in brackets, population for follow-up	Source of information on smoking (proportion of respondents)	Duration of follow-up and No. of deaths	Completeness of follow-up for mortality
American Cancer Society Nine-State Study	1952	204 547 men [187 783]	Self-administered questionnaire	44 months 11 870 deaths	98.9%
Canadian Study	1955-1956	207 397 subjects (aged 30+) [92 000]	Self-administered questionnaire (57% respondents)	6 years 9491 deaths in men; 1794 deaths in women	NA
British Doctors Study	1951	34 440 men (aged 20+)	Self-administered questionnaire (69% respondents)	20 years 10 072 deaths	99.7%
American Cancer Society 25-State	1959-1960	1 078 894 subjects First follow-up 440 558 men, 562 671 women (aged 35-84); second follow-up 358 422 men, 483 519 women	Self-administered questionnaire	4.5+5 years 26 448 deaths in men; 16 773 deaths in women	97.4% in women 97.9% in men in first follow-up
US Veterans Study	1954	293 958 men (aged 31-84) [248 046]	Self-administered questionnaire (85% respondents)	16 years 107 563 deaths	'Almost 100%' ascertainment of vital status; 97.6% of death certificates retrieved

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Figure - 2 (Contd.)

Study	Year of enrollment	Sample size: initial samples; in brackets, population for follow-up	Source of information on smoking (proportion of respondents)	Duration of follow-up and No. of deaths	Completeness of follow-up for mortality
Californian Study	1954-1957	68 153 men (aged 35-64)	Self-administered questionnaire	5-8 years 4706 deaths	NA
Swedish Study	1963	27 342 men, 27 732 women (aged 18-69)	Self-administered questionnaire (89% respondents)	10 years 5655 deaths (2968 autopsies)	NA
Japanese Study	1965	122 261 men, 142 857 women (aged 40+)	Interview (95% of population in area)	16 years 51 422 deaths	Total

NA, not available

(IARC Monograph. Vol. 38, 1986)

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Figure - 3

Royal College of Physicians Report on Smoking and Health, 1971

Expected and observed Deaths from various causes, and mortality ratios

	Expected	Observed	D	Mortality Ratio
Cancer of the lung	170	1,833	1,657	10.8
Bronchitis and emphysema	90	546	456	6.1
Cancer of the larynx	14	75	61	5.4
Cancer of the oral cavity	37	152	115	4.1
Cancer of the oesophagus	34	113	79	3.4
Stomach & duodenal ulcers	105	294	189	2.8
Other circulatory diseases	254	649	395	2.6
Cirrhosis of liver	169	379	210	2.2
Cancer of bladder	112	216	104	1.9
Coronary artery disease	6,431	11,177	4,746	1.7
Other heart diseases	526	868	342	1.7
Hypertensive heart disease	409	631	222	1.5
General arteriosclerosis	211	310	99	1.5
Cancer of kidney	79	120	41	1.5
All other cancer	1,061	1,524	463	1.4
Influenza, pneumonia	303	415	112	1.4
All other causes	1,509	1,946	437	1.3
Cerebral vascular lesions	1,462	1,844	382	1.3
Cancer of prostate	253	318	65	1.3
Accidents,suicides,violence	1,063	1,310	247	1.2
Nephritis	156	173	17	1.1
Rheumatic heart disease	291	309	18	1.1
Cancer of rectum	208	213	5	1.0

Expected = non smokers

Observed = Smokers

D = Observed - Expected

Mortality ratio = Observed ÷ Expected

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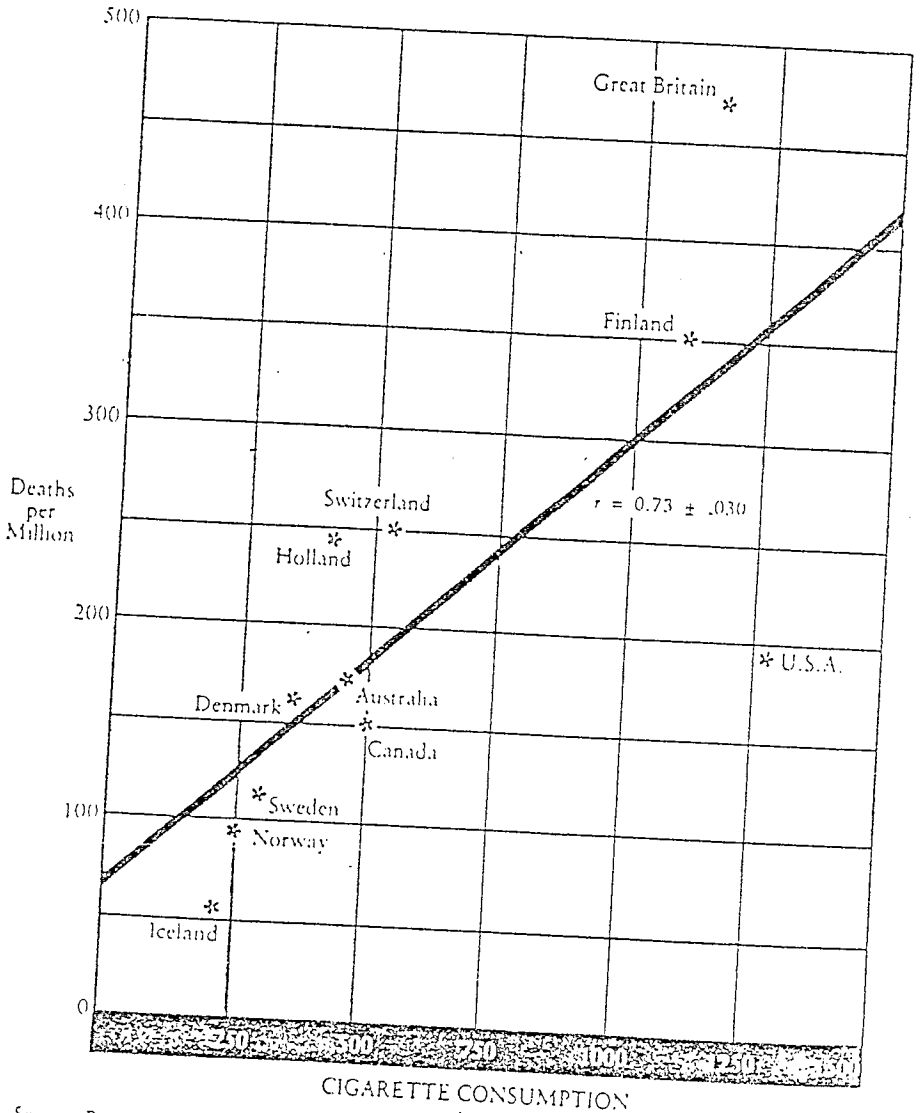
Figure - 4

Per capita consumption of cigarette and death



Source: Royal College of Physicians Report on Smoking and Health, 1971.

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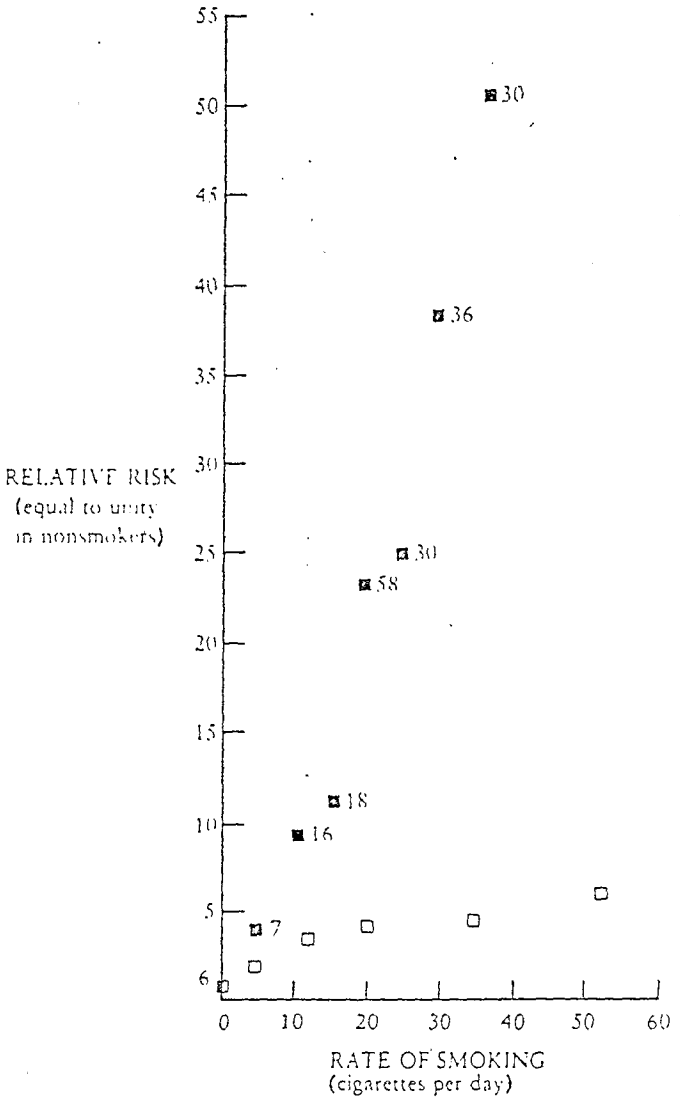
Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Figure - 5

Rate of smoking and relative risk



British male doctors
Japanese males

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Criticism of researches

The foregoing conclusions have been criticised from many view points alleging selection bias, faulty samples and lack of rigorous scientific standards in laboratory experiments apart from failure to take into consideration variables other than tobacco, like occupational history, dietary habit, personality, stress, constitutional or genetic factors. Critics have pointed out general inconsistencies, errors and unsupported conclusions to make it possible to accept the suggestion as proven that in a meaningful way tobacco causes lung cancer or cardiovascular diseases.

(In one of his reports the Surgeon General has himself stated that the seven major surveys used for his report were not designed to represent the U.S. population, because statisical methods could not establish proof.)

Dr. Dijkstra mentions that only 65% of the doctors answered the questionnaire on the survey on doctors by the Royal College of Physicians and was thus unrepresentative of the universe.

Burch pointed out that "we cannot assume that British male doctors, particularly the self-selected group that answered the Doll-Hill questionnaire, were representative of all British males". Dr. Hill admitted that the epidemiological study was not done as well as he could have wished, which prompted Burch to ask, "if an epidemiological study cannot be properly conducted, should it be done at all ?"

Sir Ronald Fisher, late of Cambridge University said "the theory that tobacco caused lung cancer, will eventually be regarded as catastrophic and conspicious howler".

Burch (Journal of Royal Statistical Society A. 1978) stated that lung cancer is due to spontaneous mutations in the tissue cells and not smoking.

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In U.S. Congressional Record, Professor M. B. Rosenblatt of New York Medical College said, "It is fanciful extrapolation not factual data". He also said, "The unscientific way in which the study was made bothers us most. The U.S. Surgeon General's committee agreed first that smoking causes lung cancer and then they set out to prove it statistically".

Even the Royal College of Physicians have admitted that only a minority of heavy smokers get lung cancer and most smokers suffer no impairment of health or shortening of life.

A study by Cooke et al (Medical Journal of Australia,January 23 1982) investigating the effects of alcohol on blood pressure in 13,000 men and 7,000 women, found that the greater the alcohol intake the higher the blood pressure, but the blood pressure levels of smokers were lower than those of non-smokers.

Professor Sheldon Sommers, told a U.S. Congressional Inquiry in 1982, that lung cancer remained a medical mystery and could not be directly linked biologically to cigarette smoking. Biomedical experimentation does not support that smoking caused cancer".

He was supported by other well-known scientists including Victor Buhler, Past President of the College of American Pathologists, Jack Matthews Farris, Professor Emeritus, University of California, San Diego, Eleanor J. Macdonald, and Lawrence L. Kupper, a bio-statistician specialising in Epidemiology and Environmental Health, University of North Carolina. All told U.S. Congressmen they did not accept the smoking-causation theory of lung cancer.

Emeritus Professor H. Russell Fisher, University of Southern California, told U.S. Congressmen that, "We just don't know

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the cause of lung cancer despite a mountainous accumulation of research". He drew their attention to a study published in the Journal of the National Cancer Institute of April 1979 which challenged that smoking was the major cause of lung cancer showing, in White females who had never smoked the increase in lung cancer in the past 40 years to be the same as for those who smoked cigarettes.

A 1985 report from the Microbiological Laboratory at Bethesda stated that in a nine-year study, over 10,000 mice of a special breed that is particularly susceptible to lung cancer were made to inhale cigarette smoke. No mice developed squamous cell lung cancer which is the type that is claimed to occur in humans upon smoking. Some mice developed other types of cancer but the incidence was the same as in the control mice that did not inhale smoke.

At a U.S. Congressional hearing in 1982, Dr. A. Furst, Director Emeritus, Institute of Chemical Biology, University of San Francisco, gave testimony that he had tried for many years to induce lung cancer in animals with cigarette smoke but without success.

Marks and Emerson and Handley and Teather (British Medical Journal, 1974) showed that after a surgical operation there was a higher risk of thrombosis in non-smokers than in smokers. This was confirmed by a report from A.V. Pollock.

Between 1951 and 1976 the death rate for heart disease in Switzerland fell by 13 per cent for men and 40 per cent for women. But sale of cigarettes was steady.

Professsor Sterling (Medical Journal of Australia, October 15, 1977) claimed that smokers get less heart diseases and refers to a study by the U.S. National Centre for Health Studies (1967) showing that non-smokers get a lot more that smokers. Rates per 100 were :

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Figure - 6

	Never Smoked	Half a pack a day	Half to one pack
Men	4.6	3.2	3.4
Women	5.5	2.0	2.2

(U.S. Public Health Services Publication No.1000 Series 10 No. 24, 1967)

Professor Schievelbein of the German Heart Centre and consultant to W.H.O. (Preventive Medicine, May 1973) said, "assuming that a major part in the etiology of cardiovascular disease is the development of arteriosclerosis, investigation in this direction has been performed on carbon monoxide and nicotine in animal experiments. Neither substance has any influence comparable to human arteriosclerosis on the development of cardiovascular disease".

In Preventive Medicine, May 1979 he also reported that tobacco smoke exposure in animals has never produced an arteriosclerotic condition similar to human disease.

These criticism include those on the alleged harmful effects of passive smoking (ETS).

The Royal College of Physicians (1977) admitted that "there was no clear evidence of damage to health from usual social exposure of other people's smoke."

At a U.S. Congressional hearing (1978) eighteen scientists gave evidence that passive smoking could cause harm.

Kerrebjin (Acta Paed Scand 106(4), 1977), after a five-year study of children concluded, "Smoking and non-smoking parents have about the same proportion of children with respiratory systoms. The number of cigarettes smoked has no influence on respiratory symptoms in their children.

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Professor H. Shievebein (Mun. Med. WSCHR 121, 1979) concluded that there was no evidence of a threat of passive smoke to health. Professor Aviado (University of Pennsylvania) said, "From the measurement of carbon monoxide levels indoors and nicotine absorbed by smokers, we can conclude that smoking in public places does not constitute a health hazard to non-smokers". Professor Klosterkotter (University of Essen) said it was "definitely impossible" for passive smoking to impair health. Professors Hinds and First of Harvard University said (1975) the alleged danger was "out of the question".

Dr. P. Harke (1970) carried out an experiment in which 150 cigarettes were smoked on a machine in a room 25 by 30 by 8 feet. He found no harmful levels.

Hans J. Eysenck has been a major critic of the methodology of most of the studies on the subject and pointed out that they were often partial observational interpretation by means of correspondennce rule, commonly referred to as the received view of theories-a causal interpretation of the statistical association between tobacco and tobacco related diseases.

This association has given rise to the speculation about the number of lives that could be saved if the use of tobacco was discountinued. (The Surgeon General Report (1982) stated that 85% of lung cancer cases were due to tobacco smoking and consequently this mortality could have been avoided if an individual never took to smoking).

This led to construction of mortality ratios,(age-standardised mortality rate, or SMR) indicating the proportion of smokers to non-smokers who are certified as having died of a particular disease (Figure 3).

Eysenck has pointed out such mortality ratio (also known as risk ratio) would differ considerably from one population

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to another and only illustrates what happened in a particular year, in a particular country. For different population at different times the ratios would give different results.

Second, the mortality ratios for smoking may be below 1 as well as above (thus apparently indicating even a beneficient effect of smoking if one were to use the received view). Khan (1966) found a mortality ratio of 0.90 for cancer of the rectum. Hammond (1966) found ratios of 0.78 and 0.66 respectively for colorectal cancer in women who smoked or smoked heavily. Choi, Schumann and Gullen (1970) found a negative correlation of primary central nervous system neoplasms with cigarette smoking.

It is not claimed that these figures indicate a beneficial effect of smoking. But, a positive mortality ratio does not necessarily indicate a harmful effect of smoking, the problem of inferring causation from correlation being more complex than that.

Third, in the case of tobacco-related cancer even if smoking were a cause of cancer, it was neither a necessary nor a sufficient cause. For Causasian groups at least, only one out of ten heavy smokers died of lung cancer. Hence smoking was not a sufficient cause. Of 10 people who die of lung cancer, one is a non-smoker. Hence smoking was not a necessary cause.

Referring to Figure-4, the UK RCP pointed out that the co-relationship the crude male death rate for lung cancer in 1950 and per capita consumption of cigarettes in 1930 (a difference of 20 years being used) was 0.73 ± 0.30.

There were many similar statistics linking diseases with external variables where a direct causal relationship is often difficult to support. There was a close correlation between consumption of meat and intestinal cancer over 22

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countries (Eysenck, 1980). Another correlation (r = 0.75 over 43 countries) between ischaemic heart disease and daily milk consumption and a correlation of 0.75 between consumption of refined sugar and ischaemic heart disease, also over 43 countries. A close relationship between sugar and breast cancer had been reported across 20 countries (Monitor, 1983; Seely and Horrabin, 1983). Following through the argument on which the received view is based, we may consider how many lives would be saved if we gave up eating meat, drinking milk and using sugar, and if these items should be given up in view of such diseases.

A third argument for the received view has been, birth cohorts have increasing age-specific mortality range with increasing smoking prevalence, implying a correlation between the amount of smoking and incidence of lung cancer. Over the periods of time considered, there has been not only increased smoking but also in the presence and number of external variables. He also points out that the mortality ratios for people who have stopped smoking for 1-4 years appear to be greater than for current smokers.

Animal tests are usually a reliable method of testing causal factors of a disease. In this connection Surgeon General's Report (1982 page 89) says that the animal models for experimental study of oral carcinogenesis have not been found to produce malignancies when cigarette smoke or cigarette smoke condensate was applied to the oral cavities of mice, rabbits or hamster.

These having been so, what do the statistical associations disclose regarding causal relations ? Brownlee (1965) in a review in the Journal of American Statistical Association concluded that the Surgeon General's Committee have not established the case for causality between smoking and lung cancer, arguing that it was not possible at that stage to reach definite conclusions, since among other thing the genetic hypothesis had not been disproved.

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If genetic factors were at all important, with the evidence indicating that they were, all arguments from correlation have to be subjected to the criticism that self selection has played an important part in the Surgeon General's reports, vitiating the observed relationship to an unknown extent. The application of statistical criteria of significance demands a process of randomisation and this is conspicuously missing in the data usually produced to support the received view.

Conclusions drawn from a statistical study of epidemiological data, are dependent not only on the logic of experimental design, and quality of statistical analysis but more crucially on the quality of actual data collected. In this regard, inaccurate or unreliable diagnosis on death certificates were important variables, Britton (1974) found that the frequency of disagreements between clinical and autopsy diagnosis ranged from 6 to 65%. Cameron and McGoogan concluded that "carcinoma of bronchus was the most common neoplasm in our series and provided the largest group of mis-diagnoses". For cardio-respiratory conditions, for acute myocardiac infarction there was agreement on 198 cases and disagreement on 109 cases, for cerebrovascular agreement on 129 cases and disagreement on 118 cases according to Cameron amd McGoogan, Heart-Vite (1979) and Heasman and Lipworth (1967).

Feinstein and Wells (1974) have published data warning of the phenomenon of "detection bias", a tendency to diagnose smoking-related diseases in smokers than in non-smokers.

The Surgeon General (1982) gave 5 criteria for evaluating causal significance of association :

Consistency
Strength
Specificity
Temporal relationship
Coherence.

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Consistency

With reference to Fig.5 Burch, (1984) considered a significant contrast in the forms of dose-response relationship.

With ratios showing ranges overall of more than two orders of magnitude, it is not evident that any acceptable criterion of consistency has been satisfied.

Strength

It is almost impossible to consider strength and consistency in isolation. The risk ratio provides only an evaluation of the importance of a factor in the production of a disease once its causal effect has been proved. It cannot be used by itself, to prove the causal relationship. If the strength of the association be found to differ from one population to another, it may provide evidence in favour of a genetic as opposed to a causal inference.

On the hypothesis of the universal causal effect of smoking on lung cancer, similar or identical ratios should be found say in Oriental and Caucasian polutation.

This, however, is not so, The causal hypothesis in its pure form, should predict the same response from the same dose in different populations. The genetic hypothesis on the other hand depended on the strength of the association between smoking and patients presenting genotypes.

The age-standardized mortality from lung cancer in Finish men in 1960-61, was double that in US white males, whereas cigarette consumption in 1950 in Finland was about half that in the United States (Burch, 1976).

Passey (1962) doubted the existence of a proper dose - response relationship within a population, mentioning that the amount smoked made no appreciable difference to the

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mean age at which the person first reported to the clinic. The light smoker is affected with lung cancer at the same age as heavy smoker.

Pike and Doll (1965) working on their sample of British doctors concluded that neither the amount smoked nor the age of starting made any substantial difference to the (period) average age of onset of the disease.

Fisher (1959) analysed retrospective data of disease by daily rate of smoking and inhalation status, and found that within a given range of smoking, inhalers had, paradoxically, a lower risk of lung cancer than non-halers.

Specificity

The existence of negative association between smoking and many of diseases makes the received view difficult to accept.

Temporal relation

The increase of cancer in the population happens on account of the increase in age which takes place at the same time in the populations in quention. When corrected for differential age distribution the increase vanishes, there being no evidence of an increase in the death rate of cancer corresponding to increased smoking. The U.K. where about 10% fewer cigarettes were smoked than in USA in 1930 had a death rate from lung cancer about 2.5 times as large in 1950, in the USA.

Coherence

The Multiple Risk Factor Intervention Priority Research Group (1982) study of groups which were not self-selected

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but included some randomisation on 12,866 high risk men aged 35-47 showed that the effect of significant lowering of consumption of cigarette had little impact on hypertension, choloestrol levels and mortality, the effect of lower consumption of cigarette being practically non-existent.

Eysenck points out that results did not disprove the possibility that smoking may cause lung and other cancer, but the evidence equally certainly does not establish that it does so and that alternative theories like constitutional or genetic hypothesis, personality, stress factors, must be considered.

Seltzer (1975) working on a population of 65,086 White, Black and Oriental smokers and non-smokers, 20-79 years of age tried to quantify the pulmonary function. For the White group, larger mean values were found among non-smokers in comparison with smokers in all age groups and for both sectors. For Blacks and Orientals no such difference between smokers and non-smokers was noted.

Large-scale studies of Japanese men by Kagan et al. 1975 onwards showed a gradient of CHD mortality, increasing from Japan to California although the rates of smoking of these men were not substantially different.

Researches on mortality due to Angina pectoris in a large scale study showed that those who smoked 20 or more cigarettes per day had a lower morbidity than non-smokers, suggesting that the risk of Angina pectoris as a sole initial manifestation of CHD, appears to be unrelated to the tobacco habit.

In a multinational study, Keys (1962) found no association between cigarette smoking and CHD in Finland the Netherlands, Yugoslavia, Italy, Greece and Japan.

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Seltzer (1968) concluded that the duration of cigarette smoking had no correlation with CHD in terms of evidence upto 1984.

The authors of the Framingham Heart study suggested that coronary heart diseases was only acutely connected with cigarette smoking not chronically, acting by the tiggering of the lethal arrhythmia or thrombosis, in subjects predisposed by an already compromised coronary circulation. It this be true the effect of smoking would be limited solely to those persons with an already diseased circulation. Such a conclusion would seem to suggest that cigarette smoking is without deleterious effect on those with a normal, healthy coronary apparatus.

In conclusion, while there was suggestive evidence, it was far from conclusive as far as the causal role of cigarette smoking in lung cancer and CHD.

Eysenck presents the constitutional and genetic hypothesis as an alternative explanation of diseases which are now being termed as tobacco-related.

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Carl C. Seltzer (Harvard University, 1988) says that results for cigarette smoking and CHD in the Framingham Heart Study do not agree with the conventional "established wisdom" on this subject. Probably the best representatiion of the "conventional wisdom" is the published statement of the U.S. Surgeon General in the 1983 report entitled "Cardiovascular Disease". Subsequent reports by the Surgeon General have added little to the 1983 statements.

The overall conclusion of the 1983 report is that "Cigarette smoking is a major cause of coronary heart disease in the United States for both men and women." The "major findings" offered as support for this conclusion are statements that: (1) in men, the incidence of CHD is twofold greater in cigarette smokers than in nonsmokers, and fourfold greater in heavy smokers; (2) in women, the rates of CHD are lower than in men, but are commensurately higher when the smoking patters are similar to those of men; (3) the risk of developing CHD increases with the duration (in years) of cigarette smoking; and (4) the cessation of smoking leads to CHD death rates that are substantially lower in the stopped smokers that in continuing smokers, and after 10 years of non-smoking, the CHD rates of former light smokers approximate those of nonsmokers.

Comparison of Surgeon General's CHD findings and those of Framingham data with respect to cigarette smoking

Criteria	Surgeon General's findings	Framingham data findings
Men		
univariate association	Strong	Weak
multivariate association	Strong	Non-significant; absent
Women :		
univariate association	Present, but less than in men	Absent
multivariate association	Present	Absent
Duration of cigarette smoking	Increase of CHD with increase of duration of smoking	Absent
Cessation of cigarette smoking	Gradual reduction CHD to level of nonsmoker after 10 years	Immediate reduction of CHD to level below that of never smoker

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

The table here compares the Surgeon General's conclusion with those that emerge from the published Framingham Heart Study data.

For men, univariate association between cigarette smoking and CHD is reported as strong by the Surgeon General, but is weak in the Framingham data. The Surgeon General notes a fourfold greater CHD incidence in "heavy" smokers over nonsmokers, but the Framingham data report relative risk ratios less than two. For multivariable analyses (involving confounding factors) the Surgeon General states that the independent effect of cigarette smoking on CHD is independent and strong, but the Framingham data, on the other hand, do not support this contention.

With respect to women, the Surgeon General asserts that cigarette smoking has a definite association and an independent effect on CHD, but the Framingham data find no association or independent effect.

For both men and women, this Framingham data do not agree with the Surgeon General's conclusion that the risk of developing CHD increases with the total duration of smoking.

The Surgeon General and the Framingham investigators agree that cessation of cigarette smoking reduces CHD and CHD death rates. The Surgeon General, however, states that the rate of CHD is gradually reduced in ex-smokers, eventually reaching a level between those of continuing smokers and nonsmokers, and sometimes (after many years) falling to the level of non-smokers. The Framingham ex-smoker data, on the other hand, show immediate, prompt, reductions to CHD rates below the level of never smokers, thus raising the strong possibility of selection bias as the main explanation for results.

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

C. Schairer et al. (Racial difference in bladder cancer risk: a case-control study. Am. J. Epidemiol. 128: 1027-1037, 1988), mentioned that racial factors are considered to be highly significant. The authors claim that while populations in the United States have approximately twice the risk of bladder cancer as black populations,and conclude that ..... "if bladder cancer incidence among whites of both sexes was reduced to the level among blacks, total incidence in the United States would fall by 36 per cent".

J. J. Gysenck (Personality, stress and cancer; prediction and prophylaxis. Br. J. Med. Psychol. 61: 57-75, 1988) summarises results from three prospective epidemiological studies in which personality inventories and other lifestyle factors were correlated with cause of death. The author claims that personality factors were more predictive of death from cancer or heart disease than was smoking, and in particular, ways of coping with stress. Stressed groups had a 40 per cent higher death rate than non-stressed groups.

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

WHO findings

(a) Chemistry of tobacco smoke

Tobacco smoke contains more than 3800 constituents, about 43 that are pharmacologically active, toxic, mutagenic and carinogenic.

However, WHO state that laboratory models for the production of diseases due to smoking are not satisfactory for diseases other than cancer. It has not been possible to show whether the particulate or the vapour phase is more important in the production of vascular and chronic obstructive lung disease. Nor do WHO know whether the nicotine content, which is generally thought to be the component of smoke that habituated smokers seek to obtain, plays any part in the production of any of the major smoking-induced diseases. (WHO, IARC Monograph on tobacco smoking, page 42, Vol. 38, 1986).

The chemical composition of smoke depends on (a) the type of tobacco; (b) design, including filtration, blend selection (e.g. reconstituted sheet, expanded tobacco), ventilation, paper and additives and (c) the smoking pattern, in the case of cigarettes.

Tobacco is smoked principally in cigarettes, with pipes, bidis and other forms being used either to a minor extent or only in certain regions. Combustion of tobacco products delivers mainstream and sidestream smoke which differ in physicochemical nature. WHO claim that sidestream smoke (SS) contains greater amounts of identified carcinogens than mainstream smoke (MS). Passive smoking is a universal phenomenon where smoking is common. The uptake of smoke constitutents by smokers and by passive smokers has been studied in only a few countries, although extensive analysis of smoke shows cigarette smoking to be a major source of exposure to tobacco-specific nitroso compounds, polynuclear aromatic compounds, aromatic amines and some carcinogens.

According to WHO smokers of cigarettes with low 'tar' yields tend to inhale to a greater extent than do smokers of cigarettes

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

with high 'tar' yields, but, in general, their intake of smoke components is reduced.

Certain biochemical markers of smoke intake, e.g., cotinine in plasma, urine or saliva, are sufficiently sensitive and specific to identify passive smokers. Passive smokers who have been examined in western Europe and North America generally have levels between about 0.1% to 1% of these markers as compared to active smokers. The precise quantitative relationship between the measured levels of these markers and the intake of carcinogenic compounds in tobacco smoke is not known.

Approximately 80% of inhaled particles from cigarette mainstream smoke is deposited in the respiratory tract, the majority in the tracheobronchial region. Wide variation is found, however, among individuals. The distribution of particulate matter in the lung is similar in smokers of 'high-' and 'low-tar' cigarettes. The pattern of deposition of sidestream smoke is very different: the proportion deposited is smaller and is likely to occur mainly in the periphery of the lung.

Regarding genetic host factors, genetic polymorphism in microsomal monooxygenases exists in humans. Lung cancer patients with a history of smoking are often extensive metabolizers of the drug debrisoquine or have high induced levels of aryl hydrocarbon hydrooxylase than smokers without lung cancer. It remains to be established whether this association implies that individuals with such genotypes are at increased risk of tobacco smoke-associated cancer.

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Figure - 7

Sales-weighted average tar deliveries in various countries, 1982[a]

Tar yield (mg/cigarette)	Developed countries	Developing countries or territories
0-10	None	None
11-16	Australia Belgium Canada Finland Germany, Federal Republic of Netherlands New Zealand Sweden Switzerland UK USA	Chile El Salvador Fiji Guatemala Kenya Mauritius Nicaragus Panama Papua New Guinea Trinidad and Tobago Venezuela
17-22	France Italy	Argentina Bangladesh Barbados Brazil Costa Rica Cyprus Hong Kong India Malawi Malaysia Malta Mexico Nigeria Sierra Leone Singapore South Africa Sri Lanka Suriname Zimbabwe
23-28	Denmark	Indonesia Pakistan Zaire
29+	None	None

[a]As supplied by the tobacco industry; from Lee, P.N. (1984)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

WHO findings

(b) Effect of Smoking

WHO state (IARC Monograph, Vol. 38, page 39, 1986) that the conclusion that smoking is one of the factors that contribute to the production or their lethality is more fairly described as presumptive than as proved, except for its association with cancer of the lung, myocardial infarction, peripheral vascular disease and chronic obstructive lung disease.

Acording to them, it was possible to distinguish with a fair degree of confidence between the disease and cause of death that are associated with smoking in different ways, as shown in figure below.

Figure – 8

Importance of different causes of death related to smoking in different ways

Category	Cause of death
A	Cancer of lung Ischaemic heart disease Respiratory heart disease Aortic aneurysm Peripheral vascular disease Chronic obstructive lung disease
B	Alcoholism Cirrhosis of liver Poisoning Suicide
C	Cancer of oesophagus Cancer of lip, tongue, mouth, pharynx, larynx Cancer of stomach Cancer of liver Cancer of bladder Cancer of kidney Cancer of pancreas Cancer of cervix uteri Cancer of unspecified site Respiratory tuberculosis Pneumonia Other respiratory disease Myocardial degeneration Hypertension Arteriosclerosis Cerebral thrombosis Other cerebrovascular disease Gastric ulcer

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Figure - 8 (Contd.)

Category	Cause of death
	Duodenal ulcer Hernia
D	Cancer of endometrium Parkinsonism Ulcerative colitis Toxaemia of pregnancy Osteoporosis
E	All others

A. Association reflecting causation.

Evidence suggest that the whole of the difference in mortality between smokers and life-long non-smokers is due to tobacco for six diseases, listed in part A, three of which are among the most common causes for death in men in developed countries.

B. Association reflecting confounding.

The difference in mortality between smokers and nonsmokers for a few other casuses has been attributed by WHO to confounding. Where smoking is presumably confounded with use of alcohol, with personality or with psychological stress, or even with all three, these are listed in part B.

C. Positive association of uncertain character.

Twenty other diseases (or group of diseases) that have been associated with smoking are listed by WHO in part C.

A variety of reasons have been suggested for their association with smoking (e.g. Doll & Peto, 1976). For some, the excess mortality in smokers is relatively small (although sometimes absolutely large) and it may be perhaps attributed to smoking on analogy with a disease

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

that smoking is known to cause. On these grounds, for example, some deaths from cerebral thrombosis may be attributed to smoking, because of the known effect of smoking on the development of atheroma and some deaths attributed to myocardial degeneration, an undiagnosed myocardial infarction. For others the evidence is confused, says WHO.

According to them other diseases, such as respiratory tuberculosis and inguinal hernia, can hardly be said to be caused by tobacco in the ordinary sense.

D. Associations possibly reflecting protection.

A few diseases that some component of tobacco smoke may help to prevent or ameliorate are listed by WHO in part D.

E. Diseases generally unrelated to smoking.

There remain other diseases that are essentially unaffected by smoking.

F. Effects on foetuses

According to WHO, smoking by pregnant women has also been found to affect foetuses transplancentally.

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WHO findings

(c) Morbility and mortality attributable to smoking

The proportion of illness attributable to smoking varies from country to country as a result of variation in the amount of illness due to other causes, the prevalence of etiological agents that interact with tobacco smoke in the production of disease, and the smoking population etc. In countries where infectious diseases are common, particularly tropical countries, where a great deal of illness is due to parasitic infection, the consumption of tobacco tends to have been small in the past, and therefore, the proportion of all illness that is now attributed to smoking is also small, say the WHO.

In other countries, according to WHO, smoking may be common and may have been so for many years, yet the effects on the incidence of some diseases may vary greatly, because of variation in the prevalence of other agents with whom it interacts. It interacts, for example, with the level of blood cholesterol, approximately multiplying the effect of high levels (Mann et al, 1976); and, on myocardial infarction it produces, although still absolutely large, can be relatively small. This is so, for example, in Japan and some other parts of eastern Asia, where low levels of blood cholesterol are prevalent due to a diet that includes relatively little fat, much of which is polyunsaturated. How far smoking interacts with other factors to increase the risk of many other smoking-related diseases is still unclear. It is, for example, uncertain how far the effect of smoke in producing chronic obstructive lung disease is modified by bacground levels of atmosphere pollution.

The third factor, the variation in smoking habits is obviously important, as the amount of disease produced by smoking cannot be large when smoking is uncommon. The effect is, however, much more complex than may appear at first sight, as heavy consumption is assocaited with little disease until smoking has been common for many years. The relationship between the risk of disease and the duration of smoking has been worked out most

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

clearly for cancer of the lung and chronic obstructive lung disease, and similar relationship may hold for many other smoking-induced diseases as well.

It is difficult, according to WHO, to estimate the contribution to morbility and mortality on different countries because of combination of these factors. It certainly cannot be estimated solely from knowledge of current smoking habits. Figures for each country can be determined only after a detailed enquiry.

The contribution of smoking to total mortality can be estimated by calculating the total number of deaths that would be observed if the population in question had experienced the same age and sex - specific total mortality rates as were observed in non-smokers. Such a calculation is, however, likely to be less reliable than the corresponding calculation of cancer deaths (page 238 of IARC Monograph, Vol. 38, 1986) because confounding between smokers and other etiological factors, such as alcohol consumption and psychological stress, is likely to be more important. In the study of Rogot and Mirray (1960) of a quarter of million US veterans, the nonsmokers' mortality rates were, in general, much lower than those observed for the whole group. Doll and Peto (1981) calculated that 15,286 of 36,143 deaths (more than 40%) occured prematurely as a result of smoking or of other factors associated with it, most of which were likely to have been due to smoking.

Similar calculations led the Royal College of Physicians (1983) to estimate that, in England and Wales, the number of deaths that occur prematurely as a result of smoking each year is at least 100,000 (i.e. approximately 18% of the total). As about one-fifth of all deaths in the U.K. and the USA are due to cancer deaths, which accounting for approximately 6 or 7% of all deaths (that is, a third of a fifth) constitute about one-half of the deaths from other diseases that have been caused in other ways by smoking (19% less 6 or 7%) (IARC Monograph, Vol.38, page 43, 1986).

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Figure - 9

Mortality by main tobacco related diseases (countries other than India)
Country : USA 1984

	All ages	Age 35 - 44	Age 45 - 54	Age 55 - 64	Age 65 - 74
1 All causes					
Male	1076 514	40 587	73 831	178 616	279 378
Female	962 855	21 969	43 382	108 739	197 192
2. All cancer					
Male	242 790	6 259	19 318	55 588	79 721
Female	210 702	7 987	19 040	44 467	60 020
3. Cancer of trachea bronchus & lung					
Male	82 491	1 477	7 570	23 055	30 044
Female	36 239	891	4 040	10 299	12 364
4. Cerebro-vascular diseases					
Male	61 697	1 180	2 677	6 664	15 050
Female	92 630	1 114	2 405	5 778	14 573
5. Bronchitis, Emphysema etc.					
Male	12 422	161	446	1 973	4 293
Female	7 950	169	400	1 373	2 591
6. Other respiratory					
Male	43 419	365	1 265	5 889	14 107
Female	26 126	269	893	3 467	7 589

Total population in '000

Total Male 115 029

Total Female 121 465

Per capita consumption (1984) of cigarette per adult population - 3250

Death by cancer of total population - 0.19%

Death by cancer of all causes of death - 22.2%

Death by lung cancer of all cancer death - 26.2%

Death by Lung cancer of all causes of cancer - 5.82%.

(WHO Annual Stats)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Figure - 10

Mortality by main tobacco related diseases (countries other than India)
Country : Japan 1986

	All ages	35 - 44	45 - 54	55 - 64	65 - 74
1. All causes					
Male	406 918	17 094	38 908	63 845	90 968
Female	343 702	9 509	18 705	33 940	65 440
2. All cancer					
Male	113 589	3 924	12 511	26 740	33 491
Female	78 065	4 156	8 610	14 815	21 026
3. Cancer of trachea broncus & lung					
Male	21 447	407	1 432	4 407	7 427
Female	8 088	270	629	1 362	2 565
4. Cerebro-vascular diseases					
Male	61 832	1 630	4 699	7 670	13 826
Female	67 457	885	2 465	4 753	12 302
5. Bronchitis, Emphysema,etc.					
Male	8 999	125	263	713	2 088
Female	5 100	76	206	390	988
6. Other respiratory					
Male	6 594	115	287	824	1 851
Female	3 558	53	161	326	805

Total population in '000

Total Male 59 438

Total Female 61 508

Per capita consumption (1986) of cigarette per adult population - 3210

Death by cancer of total population - 0.16%

Death by cancer of all cause, of death - 25.5%

Death by lung cancer of all cancer death - 15.4%

Death by lung cancer of all causes of death - 3.9%.

(WHO Annual Stats)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Figure -11

Mortality by main tobacco related diseases (countries other than India)
Country : Federal Republic of Germany 1985

	All ages	Age 35 - 44	Age 45 - 54	Age 55 - 64	Age 65 - 74
1. All causes					
Male	334 382	9 750	26 777	49 371	77 556
Female	369 914	5 058	12 540	29 995	67 031
2. All cancer					
Male	81 128	2 100	7 655	15 819	22 128
Female	81 397	2 175	5. 905	13 200	20 288
3. Cancer of trachea broncus & lung					
Male	21 707	428	2 191	5 415	6 786
Female	4 560	99	337	960	1 434
4. Cerebro-vascular diseases					
Male	34 696	577	1 853	3 784	6 638
Female	53 740	217	633	2 221	6 908
5. Bronchitis, Emphysema,etc					
Male.	14 933	74	401	1 663	3 769
Female	7 580	54	211	622	1 542
6. Other respiratory					
Male	2 717	28	117	329	682
Female	1 962	23	59	150	369

Total population in '000

Total Male 29 212

Total Female 31 812

Per capita consumption (1985) of cigarette per adult population - 2320

Death by cancer of total population - 0.27%

Death by cancer of all causes of death - 23.1%

Death by lung cancer of all cancer death - 16.1%

Death by lung cancer of all causes of death - 3.7%.

(WHO Annual Stats)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Figure - 12

Mortality by main tobacco related diseases (countries other than India)

Country : France 1985

	Age				
	All ages	35 - 44	45 - 54	55 - 64	65 - 74
1. All causes					
Male	286 892	10 065	21 945	46 255	57 315
Female	265 604	4 361	8 724	19 032	33 536
2. All cancer					
Male	80 246	2 108	8 198	18 992	20 503
Female	51 697	1 438	3 815	8 406	10 768
3. Cancer of trachea broncus & lung					
Male	17 042	437	1 948	4 999	4 930
Female	2 359	48	185	478	597
4. Cerebro-vascular diseases					
Male	26 130	354	843	2 475	4 817
Female	36 728	189	423	1 138	3 739
5. Bronchitis, Emphysema,etc.					
Male	5 269	70	179	524	1 002
Female	3 511	43	106	211	401
6. Other respiratory					
Male	8 312	78	246	1 027	1 797
Female	5 564	29	123	259	612

Total polulation in '000.

Total Male 26 900

Total Female 28 270

Per capita consumption (1985) of cigarette per adult population - 2220

Death by cancer of total population - 0.24%.

Death by cancer of all causes of death - 24%.

Death by lung cancer of all cancer deaths - 14.7%.

Death by lung cancer of all causes of death - 3.5%.

(WHO Annual Stats)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Figure - 13

Mortality by main tobacco related diseases (countries other than India)
Country : U K (England & Wales) 1985

	Age				
	All ages	35 - 44	45 - 54	55 - 64	65 - 74
1. All causes					
Male	292 327	5 776	14 838	45 704	85 695
Female	298 407	3 803	9 111	27 664	59 285
2. All cancer					
Male	73 484	1 338	4 214	14 913	85 328
Female	66 338	1 970	4 835	12 454	18 413
3. Cancer of trachea, broncus & lung					
Male	25 994	236	1 324	6 051	9 868
Female	9 798	139	543	2 332	2 605
4. Cerebro-vascular diseases					
Male	27 590	275	773	2 607	7 522
Female	45 629	258	611	2 199	7 199
5. Bronchitis, Emphysema,etc.					
Male	10 709	71	267	1 316	3 264
Female	5 518	73	202	728	1 338
6. Other respiratory					
Male	12 311	43	181	1 389	3 827
Female	6 501	24	161	777	1 754

Total population in '000

Total Male 24 330

Total Female 25 594

Per capita consumption (1985) of cigarette per adult population - 2150

Death by cancer of total population - 0.28%

Death by cancer of all causes of death - 23.7%

Death by lung cancer of all cancer death - 25.6%

Death by lung cancer of all causes of death - 6.06%.

(WHO Annual Stats)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

WHO findings

(d) Incidence of cancer

According to WHO new cases of total incidence of cancer in 1980 was estimated to be 6.35 million, almost equally divided between the sexes (3.25 million in males and 3.10 million in females) and in economies, 3.13 million cases in developed and 3.22 million in developing countries. In a world population of 4 450 million, this indicated an overall incidence rate, for all cancers, of 143 per 100 000 per year.

The study estimated that of all deaths, 1 out of 10 was due to cancer, 1 out of 4 in developed and 1 out of 16 in developing countries.

The risk for all of the major types of cancer is strongly related to age. Thus, crude incidence rates were considerably higher in population with large numbers of elderly persons. The percentage of the population aged 65 and over varies from less than 3% in eastern and western Africa, to over 14% in northern and western Europe. Therefore the incidence rates do not give a clear impression of the relative importance of cancer in different parts of the world. They are uniformly low in Africa, for example, because of the young population.

Distribution of cancer sites for various geographical areas or categories of socio-economic development and the percentage distribution of new cases of cancer in 1980 is given in Figure -15.

The distribution by sex of cancer (in developed countries) incidence for six leading sites of the disease is as follows :

Figure - 14

Male		Female	
Lung	10.4 %	Stomach	10.5 %
Colon-rectum	9.0 %	Breast	9.0 %
		Cervix uteri	7.3 %
		Mouth-pharynx	6.0 %

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Figure - 15

Percentage distribution of new cases of cancer in 1980
by sex and development group

Localisation	World		Developed countries		Developing countries	
	M	F	M	F	M	F
Mouth-pharynx	7.9	3.9	5.0	1.7	10.8	6.1
Oesophagus	6.2	3.5	2.5	1.1	9.9	5.8
Stomach	12.6	8.4	12.2	9.0	13.0	7.8
Colon-rectum	8.8	9.2	11.7	13.2	5.9	5.4
Liver	5.3	2.6	2.3	1.5	8.2	3.6
Pancreas	2.3	2.0	3.0	2.8	1.6	1.3
Larynx	3.2	0.5	3.0	0.4	3.5	0.7
Lung	15.8	4.7	22.3	6.3	9.4	3.2
Female breast	-	18.4	-	22.9	-	14.2
Cervix uteri	-	15.0	-	6.3	-	23.3
Corpus uteri	-	4.8	-	6.8	-	2.9
Ovary	-	4.4	-	4.6	-	4.2
Prostate	7.3	-	11.0	-	3.6	-
Bladder	5.2	1.7	7.1	2.2	3.2	1.2
Lymphoma	4.3	3.2	3.9	3.5	4.7	2.8
Leukaemia	3.3	2.6	2.8	2.4	3.7	2.8
All other sites	17.8	15.1	13.2	15.3	22.5	14.7
Total	100.0	100.0	100.0	100.0	100.0	100.0

In 1980, stomach cancer was judged to be the most common cancer in the world, estimated 669 400 new cases.

Lung cancer, estimated to be 665 000 cases in 1980 has shown a progressive increase since 1975 (about 0.5% per year for the world as a whole).

Cigarette smoking has been common in the developed countries, particularly among men, for five or six decades. Among women, incidence of lung cancer is rising in parallel with cigarette consumption. In Europe and Northern America, lung cancer now accounts for 12-18% of all new cancers, (15 - 26% according to us).

In most developing countries cigarette smoking is a nore recent phenomenon, and the young and middle-aged adults who are now heavy smokers have yet to enter the age groups at high risk for lung cancer.

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Consequently, incidence rates are at present quite low although this is likely to change in the near future. The global picture is dominated by the frequency of oral cancer in Southern Asia and in Papua New Guinea, and of oral cavity plus nasopharyngeal cancer in South Eastern Asia. Oral cancer in these areas is a result of chewing of tobacco and betel nuts.

Geographical and temporal variations in cancer incidence give important clues in the search for etiological factors, and should also be consonant with the distribution of suspected risk factors identified in other ways. Such studies are also essential in the setting of priorities for, and the planning of, cancer control programmes, and in monitoring their effectiveness.

According to WHO, lung cancer is believed to be the most important cause of death from cancer in the world, major cause being tobacco smoking, primarily of cigarettes.

Risk of lung cancer is believed to be particularly dependent on duration of smoking; therefore, the earlier the age at initiation of smoking, the greater the individual risk. Further, the longer the time period during which a major proportion of adults in a population have smoked, the greater the incidence and mortality from the disease in that population. Risk of lung cancer is also proportional to the numbers of cigarettes smoked, increasing with increasing cigarette usage. In populations with a long duration and heavy intensity of cigarette usage, the proportion of lung cancer attributable to smoking is of the order of 90%, attributable to men in most western populations.

Although cigarettes are the predominant cause of lung cancer, some increased risk also results from pipe and/or cigar smoking.

According to WHO tobacco smoking is an important cause of oral, oropharyngeal, hypopharyngeal, laryngeal and oesophageal cancers. Pipe and/or cigar smoking appears to increase the risk of these cancers to approximately the same extent as cigarette smoking.

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

The risks of these cancers associated with cigarette smoking are substantially increased in conjunction with high-dose exposure to alcohol. Tobacco smokers also appear to have increased risks for cancer of the lip.

Cigarette smoking is an important cause of pancreatic cancer and perhaps of renal adenocarcinoma. The proportion of these diseases that is attributable to smoking is not possible to quantify with the same accuracy as for lung cancer. The data now available on tobacco smoking and stomach and liver cancers do not permit a conclusion that the associations noted in some studies are causal.

Although the risk of cancer of cervix is increased in tobacco smokers, it is not possible to conclude that the association is causal. Further, although in some studies a reduction in risk of endometrial cancer has been found in smokers as compared to non-smokers it cannot be concluded that smoking protects against cancer at this site.

Although smokers of 'low-tar'-level cigarettes tend to compensate for lower yields of nicotine and perhaps other smoke components, chiefly by changing the manner of smoking, they do not in general compensate fully for lower tar yields.

Case-control and cohort studies that prolonged use of nonfilter and 'high-tar' cigarettes is associated with greater lung cancer risks than prolonged use of filter and 'low-tar' cigarettes.

According to WHO, there was sufficient evidence that inhalation of tobacco smoke as well as topical application of tobacco smoke condensate cause cancer in experimental animals. Now there is sufficient evidence that tobacco smoke was carcinogenic to humans. The occurrence of malignant tumours of the respiratory tract and of the upper digestive tract is causally related to the smoking of different forms of tobacco (cigarettes, cigars, pipe, bidis). The occurrence of malignant tumours of the bladder, renal pelvis and pancreas is causally related to smoking of cigarettes, says the WHO.

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

WHO findings

(e) Carcinogenicity studies in animals

According to WHO (IARC Monograph, Vol.38, 1986, pp 194-198) there were technical problems in the generation of smoke and its delivery to animals. Moreover, respiratory systems are different for amimals. In one study involving long-term exposure of rats to cigarette smoke, tumours of the respiratory tract were induced. In hamsters, various experiments demonstrated reproducibly the induction of laryngeal carcinomas.

Studies in rabbits and dogs of whole ciagrette smoke were inadequate for evaluation.

No treatment-related tumour other than in the respiratory tract has yet been produced by smoke inhalation in experimental animals.

Experiment on the carcinogenicity of the gaseous phase of cigarette smoke in hamsters and rats resulted in negative or inadequate findings. An increased incidence of lung tumours was observed in one study in mice.

Exposure of hamsters pretreated with 7, 12 - dimethylbenz (a) - anthracene to whole smoke resulted in a significant increase in the incidence in the tumours of the respiratory system. Such effects were less pronounced when hamsters or rats were pre-treated with Benzon-(a)-pyrene. A carcinogenic effect between radon daughters and cigarette smoke was observed in the induction of lung tumours in rats.

In some experiments, animals exposed to cigarette smoke survived longer than controls; in other experiments, rats exposed to smoke had lower incidence of some tumours (e.g., mammary tumours) than did controls.

An extensive number of studies on the carcinogenicity of cigarette-smoke condensate (CSC) on mouse skin have demonstrated consistently the induction of benign and malignant skin tumours. The carcinogenic effect would appear to result from the interaction

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

of various CSC constitutents, since CSC and its constitutents have been shown to possess tumour-initiating, tumour-promoting and other cocarcinogenic activities. Cigar and pipe smoke tars are also carcinogenic to mouse skin. The mouse-skin assay may not, however, detect all of the carcinogenic activity of whole ciagrette smoke, since CSC lacks most of the volatile and many of the semi-volatile constituents of whole smoke. Skin tumours have also been observed in rabbits following application of CSC.

Direct injection of CSC in a lipid vehicle into the lungs of rats caused squamous-cell carcinomas of the lung. Topical application of CSC to the oral mucosa of mice resulted in the induction of lung tumours and, perhaps lymphomas.

WHO findings

(f) Observations in human

A general dose-response relationship is observed between the number of cigarettes smoked and the levels of tobacco-specific intake markers such as nicotine, its metabolite cotinine and carboxyhaemoglobin according to WHO. Pipe and cigar smokers usually have lower carboxyhaemoglobin values than cigarette smokers, because they inhale less.

Several reports demonstrated increased uninary mutagenicity in smokers.

Approximately 80% of the 'tar' inhaled from mainstream cigarette smoke is deposited in the respiratory tract - the majority in the tracheobronichial region. There is wide inter-subject variation in the amount of 'tar' deposited, depending on the pattern of smoke inhalation and exhalation.

Tobacco smoke impairs lung defence mechanism and affects some systemic aspects of the immune system. The consequences of these changes on tobacco smoke pathogenesis are currently unknown.

It is uncertain whether genetically determined differences in the matabolism of some foreign substances underlie increased risks

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

of lung and bladder cancer in cigarette smokers. The differences may be secondary to malignancy rather than predisposing to its development.

Tobacco smoking results in genetic damage to somatic cells. Cigarette smokers have significantly raised levels of chromosomal damage (structural aberrations and micronuclei) in somatic cells. There is some evidence according to WHO that the prevalence of chromosomal aberrations in blood cells of smokers is a function of the number and 'tar' yield of cigarettes smoked. More positive evidence for a dose-response relationship is provided by studies on the prevalence of sister chromatid exchanges in somatic cells of smokers.

Studies on germ cells and on the products of conception of smokers provide equivocal results as to whether or not cigarette smoking results in heritable mutations that are transmitted to progeny. According to WHO the evidence concerning as increased prevalence of morphologically abnormal sperm in smokers is conflicting. More data are required before any firm conclusion can be drawn.

(IARC Monograph, Vol.38, 1986, pp 194-196)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Indian Medical Researches

Indian researches on the health effects of tobacco have not been extensive. No country-wide analysis of smoking trends has been carried out so far.

Among the few studies which have been carried out, the following may be mentioned :

The earliest observation on the relationship of cheek cancer with the habit of chewing betel leaf with tobacco was made in 1902 by W. J. Niblock of Madras General Hospital.

Orr reported an oral cancer in betel nut chewers in Travancore (now Kerala) in Lancet in 1933.

A 1955 case-control study by Sanghvi, Rao & Khanokar showed a significant role of bidi in the cancers of oral cavity, pharynx and oesophagus; like tobacco chewing habit, its major effect being on cancers of the oropharynx with relative risks being higher in persons who smoked bidi and used chewing tobacco, at the same time.

Jussawala & Despande confirmed these observations in 1971.

Notani & Sanghvi came to the conclusion in 1974, confirmed by Jussawala and Jain in 1979 that the risk of bidi in lung cancer was equally high, if not higher than cigarette and there was a dose-response relationship between its smoking and cancer.

Bhide, Kulkarni, Padma et al carried out (1989) work at the Cancer Research Institute, Tata Memorial Centre, Bombay on chewing tobacco and gave evidence of several N. nitroso compounds, being potent careinogens in N.Rustica, used mainly in chewing and hookah, with tobacco specific nitrosamines (TSNA's) higher in Rustica tobacco than N. Tabacucum.

For Bidi, chemical analysis of bidi smoke was carried out in 1974 by Hoffman, Sanghvi and Wynder at a US laboratory. It was

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found that bidi delivered a large amount of tar, nicotine and other toxic chemicals and also that, because of the less porous tendu leaf, carbon monoxide level in bidi smoke was higher in bidi than in cigarette.

Sanghvi, Jayant & Pakhale in 1980 found higher levels of tar and nicotine in Indian cigarettes than in the west.

Based on Khanolkar's 1950 study on tobacco habits, Sanghvi estimated (1989) patterns of consumption as follows :

Figure - 16

Estimated No. of adults in millions aged 15 years and over in India, with different tobacco habits

50 - 55 to 80 - 83

Tobacco habit	1950-55	1965-70	1980-83
Cigarette smoker	6	18	22
Bidi smoker	58	79	136
Tobacco chewer	47	49	31
Total population	230	300	400

(assumptions made, bidi/cigarette smokers smoke on average 10/day, chewers consume 3g/day. A cigarette on average contains 1g of tobacco, bidi 0.25g, thus a cigarette smoker smokes 3.65 kg bidi smoker 0.91 and a chewer 1.1 kg per year)

According to S P Gupta (1989) reliable data in India on the effect of tobacco on cardiovascular diseases was unavailable and consequently research data had to remain tentative.

Jindal and Malik (1989) found a relationship with cheroot smokers in Shimla hills and south-east Orissa and prevalence of chronic bronchitis and emphysema. Gupta, PC et al. in a major age-adjusted relative risk study in Ernakulum, Kerala and Srikakulam, Andhra Pradesh, over 10 years in 1980 and 1984 concluded,

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

that extra death per year attributable to the use of tobacco would be a maximum of about 630,000, going up to 1,000,000.

Notani et al. found (1989) the highest proportion of morbidity cases attributable to tobacco and therefore avoidable was in cancers of the upper alimentary and respiratory tract (63%). However since these cancers form only 1% of the total morbidity, the overall impact was limited. Next in rank came chronic obstructive lung diseases (COLD) with 45% avoidable cases and coronary heart diseases (CHD) 21% avoidable cases, the avoidable proportions being much higher in males (55%) than in females (6%).

They estimated age - sex - specific deaths from upper alimentary and respiratory tract cancer deaths to be 293,500, from CHD 450,000 and from COLD 350,000 in 1986.

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Toxicity in Indian Tobacco Types

Following toxicities have been found in various types of tobacco products in India :

a)

Figure - 17

	Bidi	Cigarette (India)
Tar	23-41 mg	19-28 mg
Nicotine	1.7-2.8 mg	0.9-1.8 mg
C.O.	7.7 vol. %	
Hydrogen cyainde	668µg to 904µg	366-638µg
Phenols	129-273µg	118-226µg
Benzo (a) pyrene	108-144 ng	85-114 ng

Notani, Jayant and Sangvi, 1991
(Cigarette, based on 1974 study)

b) Gaseous components of the cigarette smoke, not available.

c) In chewing tobacco several N-nitroso compounds have been identified.

d) 24g of hookah tobacco smoked through 150 ml of water gave total (dry) particulate matter of 9.1 mg & nicotine of 0.55 mg for a total smoking time of 35 minutes.

Age distribution

Distribution by age in years in India in 1991 was as follows :

Figure - 18

Age groups (% of total population)			
0 - 14	15-44	45-65	65 +
35.6	60.3	13.9	4.1

(1991 total population 837 million)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Age specific deaths in India (1984) — Figure - 19

(Percentage)

Age	Rural	Urban
0 - 4	46.2	23.2
45 - 49	8.5	7.7
50 - 54	13.8	12.0
55 - 59	18.3	18.4
60 - 64	33.8	31.2

Consumption in India

NSS reports (43rd round 1987/88) on prevalence of tobacco habits in India as follows :

a) Consumption of tobacco amount, rural, urban :

In rural India about 34% of the male population are found to use tobacco in some form regularly, while 24% are regular smokers. Only 10% of the female population in rural India consume tobacco regularly in any form. Of the females in rural India only 2% smoke regularly. Females, in general, show a comparatively greater inclination to consume other forms of tobacco.

In urban India 24% of males consume tobacco regularly while 18% are regular smokers. Only 5% of urban females consume tobacco, of whom only 0.7% are regular smokers. In urban India, the proportion of female casual tobacco consumers is found to be very negligible.

b) Consumption of tobacco by type :

20% and 17% of the total males are exclusive smokers in the rural and urban India. The proportion of female smokers are 2% and 0.7% in the rural and urban India. Chewing claims 8% of rural males and by 5% of urban males. For females it is 5% in rural India and 3% in urban India.

For snuff and burnt tobacco etc. the proportions of female consumers in both the rural and urban areas are higher compared to that of male consumers. For various other

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

combinations, the incidence seems to be negligible. There is predominance of smoking seen amongst males but in females other forms ot tobacco consumption were more prevalent.

c) **Consumption of tobacco by age and sex :**

It is seen from the table below that tobacco use increased with age. Males were predominantly smokers and about 50% of them in rural areas & 40% in urban areas were in the habit of smoking, after attaining 25 years of age, around 70% of the rural males and around 50% of urban males found to be consuming tobacco in any form after attaining 25 years of age. Consumption of tobacco in any form was least in the age group 0-14 years.

Figure - 20

Number of persons reporting consumption of tobacco in various forms either exclusively or in combination per 1000 persons by sex : All-India rural and urban

Forms of tobacco consumption	Rural		Urban	
	Male	Female	Male	Female
Smoking (SM)	199	21	169	7
Chewing zarda etc. (CHW)	77	47	46	28
Snuff (SN)	4	7	3	4
Burnt tobacco, powder, paste (BT)	12	26	7	14
SM and CHW	41	3	22	1
SM and SN	1	-	1	-
SM and BT	6	1	3	-
SN and BT	1	1	-	-
CHW and BT	7	5	4	3
CHW and SN	1	-	-	-
SM, CHW and SN	-	-	-	-
SM, CHW and BT	2	-	1	-
SM, SN and BT	-	-	-	-
CHW, SN and BT	-	-	-	-
SM, CHW, SN and BT	1	-	1	-

(Source : NSSO Govt. of India 43rd round 1987-88)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Figure - 21

Number of persons reporting smoking and/or consumption of tobacco in any form regularly or casually per 1000 persons by sex in Rural areas.

State/Union Territory	No. of persons reporting consumption per 1000 persons							
	Male				Female			
	Smoking		Consumption of tobacco in any form		Smoking		Consumption of tobacco in any form	
	Regularly	Casually	Regularly	Casually	Regularly	Casually	Regularly	Casually
Andhra Pradesh	342	12	355	12	54	3	103	7
Assam	279	39	352	56	23	19	99	68
Bihar	134	23	313	24	41	3	62	5
Gujarat	332	7	354	8	15	3	94	6
Haryana	338	16	349	12	38	7	48	8
Himachal Pradesh	334	10	337	10	24	2	24	2
Jammu & Kashmir	226	13	295	12	35	5	69	6
Karnataka	216	13	280	14	2	1	96	10
Kerala	251	31	284	33	5	2	88	11
Madhya Pradesh	224	15	384	10	5	1	92	4
Maharastra	120	12	378	15	5	1	242	12
Manipur	329	21	353	29	125	37	147	62
Meghalaya	381	20	396	34	104	42	213	73
Nagaland	-	-	-	-	-	-	-	-
Orissa	162	25	413	21	13	5	322	11
Punjab	96	6	111	6	1	-	2	1
Rajasthan	307	9	328	9	29	2	45	2
Sikkim	188	-	219	1	109	2	109	2
Tamil Nadu	167	13	266	17	2	1	118	9
Tripura	426	7	455	12	65	6	181	18
Uttar Pradesh	254	13	345	10	26	3	63	4
West Bengal	340	16	381	15	20	3	185	7
Andaman & Nicobar	178	28	370	32	20	6	103	24
Arunachal Pradesh	388	191	190	189	31	40	173	43
Chandigarh	345	-	375	-	27	18	45	-
Dadra &Nagar Haveli	305	4	337	11	5	-	29	3
Delhi	204	3	221	3	11	-	33	-
Goa,Daman & Diu	263	4	316	4	9	-	77	-
Lakshadweep							290	
Mizoram			500				350	
Pondicherry								
All India	240		340		20		100	

(Source : N.S.S.O. Govt. of India 43rd round 1987-88)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Figure - 22

Number of persons reporting smoking and/or consumption of tobacco in any form regularly or casually per 1000 persons by sex in Urban areas.

State/Union Territory	No. of persons reporting consumption per 1000 persons							
	Male				Female			
	Smoking		Consumption of tobacco in any form		Smoking		Consumption of tobacco in any form	
	Regularly	Casually	Regularly	Casually	Regularly	Casually	Regularly	Casually
Andhra Pradesh	215	15	223	19	16	2	48	4
Assam	225	45	310	38	5	-	39	18
Bihar	77	22	220	28	9	1	29	4
Gujarat	176	5	217	8	3	2	38	4
Haryana	249	15	265	20	16	5	21	5
Himachal Pradesh	303	5	304	5	8	-	8	-
Jammu & Kashmir	275	15	287	15	16	1	21	1
Karnataka	169	14	195	19	3	2	39	5
Kerala	231	19	250	21	5	2	46	6
Madhya Pradesh	157	23	249	20	4	-	60	3
Maharastra	103	16	235	19	3	1	111	8
Manipur	278	13	315	25	101	21	141	53
Meghalaya	302	31	352	41	16	7	129	79
Negaland	148	338	198	433	-	4	36	477
Orissa	161	31	305	40	5	2	196	17
Punjab	162	8	172	9	5	-	8	-
Rajasthan	217	6	249	6	10	1	39	2
Sikkim	139	2	169	-	52	-	64	-
Tamil Nadu	170	11	197	14	3	1	43	2
Tripura	402	22	462	22	8	3	124	27
Uttar Pradesh	192	9	236	14	10	1	32	7
West Bengal	304	17	362	13	5	1	53	8
Andaman & Nicobar	127	58	268	109	5	10	35	34
Arunachal Pradesh	62	18	139	8	13	9	40	21
Chandigarh	185	7	212	7	5	-	5	-
Dadra & Nagar Haveli	-	-	-	-	-	-	-	-
Delhi	181	18	197	13	11	-	15	-
Goa,Daman & Diu	154	11	181	11	8	-	19	4
Lakshadweep	244	28	251	47	6	-	198	39
Mizoram	397	48	484	49	206	66	378	80
Pondicherry	138	22	157	22	7	-	60	19
All India	182	15	239	18	7	1	53	6

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Figure - 23

Consumption of tobacco per 1000 persons by age and sex
All India 1987 - 88

Forms of tobacco	Rural/ Urban	Age in years 0 - 14		15 - 24		25 - 44		45 - 59		60 & above		All ages	
		M	F	M	F	M	F	M	F	M	F	M	F
Smoking	Rural	6	1	180	17	519	38	567	68	456	61	252	25
	Urban	3	-	94	4	394	11	452	27	332	28	197	8
Chewing	Rural	4	4	89	30	239	87	317	149	307	150	130	56
Zarda etc.	Urban	2	1	37	10	135	42	182	106	153	119	73	33
Snuff	Rural	1	-	5	3	13	11	19	25	20	33	8	9
	Urban	-	-	1	2	7	7	14	15	22	20	5	6
Burnt tobacco	Rural	8	8	32	34	45	50	58	60	50	51	30	33
powder/paste	Urban	4	5	14	18	25	27	35	32	20	27	16	17
Consumption	Rural	16	14	268	78	689	169	792	274	702	269	353	111
in any form	Urban	7	6	132	33	494	78	593	163	472	178	257	59

(Source : NSSO Govt. of India 34rd round 1987-88)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

d) Per-capita monthly expenditure on tobacco :

Per-capita monthly expenditure on tobacco in rural and urban areas was respectively Rs.2.9 and 3.6, which constituted 1.8% & 1.4% respectively of the total percapita expenditure, as shown in Figure 24, given below. Further per-capita expenditure on tobacco constituted 5.1% and 3.2% of the total per-capita expenditure on non-food items in rural and urban communities respectively.

Figure - 24

Per-capita monthly expenditure on tobacco
(1987-88) - All India

Item	Unit of consu-ption	Rural		Urban	
		Quantity consumed	Value (Rs.)	Quantity consumed	Value (Rs.)
Bidi	No.	50.58	1.96	40.29	1.66
Cigarette	No.	1.02	0.26	5.21	1.56
Tobacco leaf	Kg.	0.02	0.42	0.01	0.20
Snuff	gm	1.14	0.03	0.73	0.02
Others	-	-	0.23	-	0.12
Total			2.90		3.56

(Source : N.S.S.O., Govt. of India 43rd round).

Notani et al. (1980), on the basis of adult population and an estimated daily intake of 15 bidis/10 cigarettes a day, came to the conclusion that there were 115 million smokers in India, 52-50% males & 2.7-2.5% females. Gupta's ('88) projection of sex-wise consumption based on around 217 000 house to house studies gave a range of 62-82% male and 15-67% for female who consumed tobacco.

A study of ICMR indicated the prevalence of tobacco usage varying from 56-64% in males and 14-43% in females (1981-1984).

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

In a survey by ICMR of tobacco habits in Goa, it was observed that 40% of all habituates had acquired the habit by the age of 20 years. The major reasons for intiating the tobacco habit among males were influence of friends 71.4%, relieving constipation 14.1%, passing time 19-26%, as a medicine 11-16%. Among females, the major reasons were influence of friends 76.7% initating elders 19.8%. It was also observed that among females 8% gave suppression of hunger, being the reason for initiating tobacco habit (ICMR, 1988).

From 1950-55 to 1980-83, total tobacco consumption per adult per year came down from an average 900g during 1950-55 to 700g during 1980-83, with chewing, hookah, cigar, cheroot and snuff having all declined and bidi & cigarette (specially the former) registered a rise. This is shown in Figure 25.

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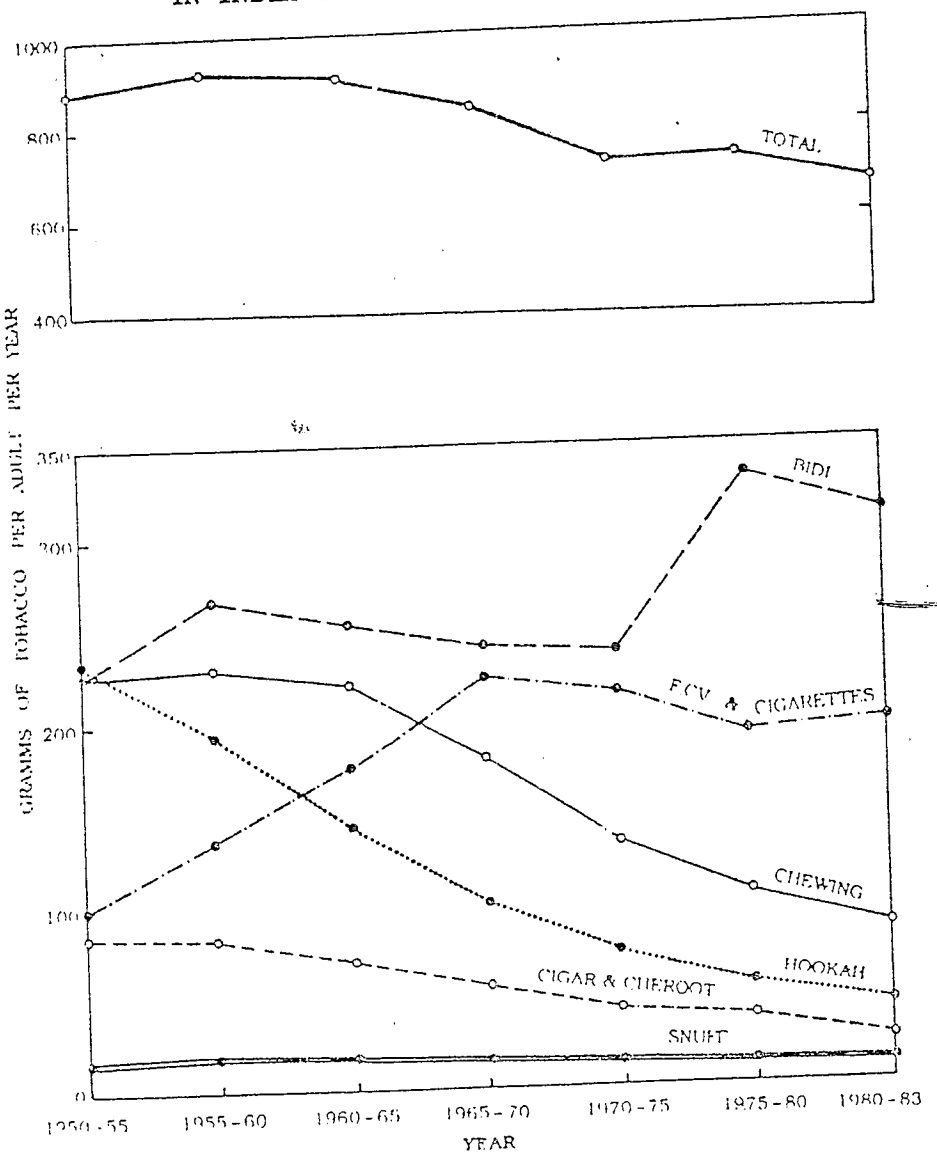
Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Figure - 25

TOBACCO CONSUMPTION IN GRAMMES PER ADULT PER YEAR
IN INDIA FROM 1950-1955 TO 1980-1983



(Sanghvi 1989)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Figure - 26

Consumption of cigarette (av.) per adult (15+) population in a few selected countries (1989)

Country	Cigarette consumption ('000 m.)	Per capita consumption	Filter %
Canada	47.6	2300	99
USA	533.0	2780	96
Brazil	162.2	1710	99
Mexico	52.0	1010	77
Belgium/Luxembourg	17.6	2070	91
France	94.9	2120	76
Germany (FRG)	118.6	2260	93
Greece	28.7	3570	95
Italy	97.4	2050	97
Spain	79.8	2560	99
UK	97.3	2100	98
Iceland	0.4	2240	NA
Switzerland	16.0	2930	97
Bulgaria	16.5	2270	
Checkoslovakia	26.4	2220	98
Hungary	26.9	3220	92
Poland	95.1	3350	NA
Romania	31.9	1790	NA
USSR	468.0	2180	NA
Yugoslavia	55.8	3060	99
Ghana	1.7	220	95
Kenya	6.2	510	68
Malawi	1.1	250	73
Bangladesh	15.2	250	41
India*	84.2	160	50
Pakistan	31.7	530	93
Sri Lanka	4.8	440	93
China	157.0	2120	42
Indonesia	16.1	1300	70
Japan	313.8	3100	99
Hongkong	7.1	1570	98
Korea (South)	91.7	2930	100
Malaysia	17.8	1660	98
Philippines	67.8	1910	91
Thailand	36.9	1010	86
Australia	34.8	2650	99
New Zealand	4.6	1770	98

* Excludes Bidi & chewing which if added, would make the figure 600 instead of 160, upon adjustment of quantity in Kg. and unit sale usage.

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Figure - 27

Trend of consumption of cigarette per adult (15+) population by countries

(No. of cigarettes)

	1970-74	China	Year	India
USSR	+ 10%	NA	1969	200
Poland	+ 14%	NA	1970	200
Hungary	+ 15%	NA	1971	190
Romania	+ 23%	NA	1972	190
Checkoslovakia	+ 32%	NA	1973	190
Bulgaria	+ 36%	NA	1974	180
Yugoslavia	+ 47%	NA	1975	190
GDR	+ 54%	840	1976	180
Average	+ 16%	890	1977	180
		900	1978	190
		990	1979	200
		1110	1980	190
		1190	1981	210
		1190	1982	220
		1330	1983	180
		1470	1984	180
		1570	1985	170
		1660	1986	170
		1760	1987	150
		2080	1988	160
		2120	1989	160

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Figure - 28

Tobacco consumption per adult per annum, by country or area

	All tobacco goods (kg)			
	1935	1950	1965	1973
*Argentina	2.0	2.1	2.0	2.2
Australia	1.8	2.9	3.4	3.4
Austria	1.7	1.5	2.2	2.7
*Barbados	-	-	1.2	1.7
Belgium	3.2	2.8	3.5	3.9
*Brazil	2.5	2.6	2.1	2.0
Canada	2.2	3.4	4.6	4.5
*Chile	-	-	1.1	1.4
*Costa Rica	-	-	1.7	1.6
Denmark	3.0	3.7	3.7	3.6
*El Salvador	-	-	1.7	1.5
Finland	1.2	1.6	2.0	2.4
France	1.7	1.9	2.4	2.7
Germany, Fed. Rep.	-	1.9	2.8	3.1
*Ghana	-	-	0.5	0.6
Greece	1.1	1.8	2.2	2.9
*Hong Kong	-	-	3.1	2.5
Iceland	1.2	2.2	3.0	3.4
8India	0.9	0.9	0.8	0.6
*Indonesia	-	-	-	0.8
Ireland	2.0	3.1	3.0	3.1
Italy	0.9	0.9	1.7	2.0
*Jamaica	-	-	1.3	1.4
Japan	1.4	1.4	2.4	3.3
*Kenya	-	-	0.4	0.4
*Malawi	-	-	0.1	0.2
*Malaysia	-	-	1.4	1.5
*Mauritius	-	-	1.3	1.5
*Mexico	1.5	1.5	1.5	1.4
*Morocco	0.4	0.6	0.7	0.8
Netherlands	3.8	3.4	4.2	4.5
New Zealand	2.0	3.3	3.4	3.2
*Nicaragua	-	-	1.0	1.2
Norway	1.4	1.9	2.1	2.4
*Pakistan	-	-	1.7	1.7
Portugal	0.6	0.9	1.2	1.5
*Sierra Leone	-	-	0.7	0.6
*Singapore	-	-	2.7	2.9
South Africa	1.5	2.2	2.1	2.3
Spain	1.6	1.4	2.0	2.5
Sweden	1.6	1.9	2.1	1.8
*Switzerland	2.1	2.7	4.1	4.3
*Turkey	1.2	1.4	2.0	4.8
United Kingdom	2.2	2.6	2.7	2.8
USA	3.5	4.6	4.9	4.2
*Venezuela	-	-	-	-

* Denotes developing countries

(Source : Report of the WHO Committee on Smoking Control, 1979)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Morbidity in India

Tobacco related diseases

Based on the estimated 1986 total population of 760 million (390 million males and 370 million females), the incidence of new cases per year of major tobacco related diseases in India was estimated by Notani et al. (1989), as follows :

a) Cancer : 270,000

b) Cardiovascular disease (CVD) in the 40-49 age group 320,000 to 448,000.

Coronary heart diseases (CHD) : 1,300,000

c) Chronic obstruction Lung disease (COLD) : 7 to 13 million

However, after examining observations of Sapru (1989) they arrived at the following conclusions :

Figure - 29

Tobacco related diseases ('000)
1989 population

	Cases		
	Male	Female	Total
Cancer (a)	233	27	270 (63%)
Cancer (b)	N.A.	N.A.	N.A.
CHD (age $\geqslant$ 30 years)	1,212	58	1,270 (21%)
Cardiovascular	N.A.	N.A.	N.A.
Peripheral vascular (age $\geqslant$ 40 years)	N.A.	N.A.	N.A.
COLD (age $\geqslant$ 15 years)	9,420	440	9,860 (45%)

(a) = upper alimentary & respiratory tracts
(b) = other tobacco related cancers
% = indicates number of tobacco related cases to the total number of cases of the disease category.

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Parkins (1984) mentioned that of the 5.8m. new cancer cases in the world, 50% were in developing countries.

Tobacco related cancers

a) Figure - 30

Tobacco related cancer in India, estimates for 1986

Site wise cancer	Male	Female	Total
Total population Millions	390	370	760
Crude rate/100,000	70	80	75
No. of new cases/year	273,000	296,000	569,000
New cases related to tobacco habits :			
Mouth	20,888	24,642	55,530
Pharynx & larynx	42,159	9,435	51,594
Oesophagus	20,280	14,874	35, 154
Lung	23,673	4,477	28,150
Others	13,000	6,572	19,572
Total	130,000 (48 %)	60,000 (20 %)	190,000 (33 %)

(Sanghvi 1989 based on 1982-1983 National Cancer Registery, 3 P/B registers, 3 H/R registers)

b) Site-wise cancer in India % wise is given in Figure -32.

c) Tata Memorial Hospital Figure - 31

1970 - 72	M	F
Lung cancer	5.7 %	0.9 %
Tongue	15.5 %	3.3 %
Mouth	10.3 %	6.6 %
Pharynx	23.6 %	4.5 %
Oesophagus	11.3 %	8.1 %
Stomach	1.7 %	1.0 %
Colon Rectum	2.9 %	1.7 %
Larynx	1.8 %	0.8 %
Breast	0.1 %	17.9 %
Cervix	-	35.5 %
Prostate	0.7 %	-
Bladder	1.1 %	0.3 %

(Jussawalla et al. 1973)

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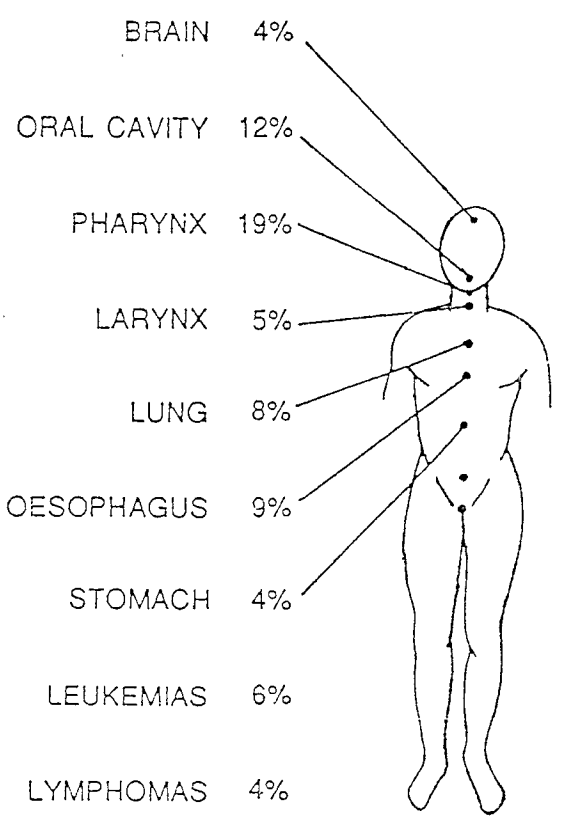
Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Figure - 32

Cancer Patient Load in Hospital Registries in India.



(Estimates based on weighted averages of data from Bangalore, Bombay, Chandigarh, Dibrugarh, Madras, Trivandrum for 1986)

(Source : National Cancer Registry)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

d) According to Madan, Dhawan & Saha, 1986, Buccal mucosa (cheek) cancer was 35% of all cancer cases :

Figure - 33

	Age group	Incidence
Cheek cancer	41 - 50	40.0 %
	51 - 60	29.1 %

	No. of cases M	F
Tobacco Chewers	72.5	72.0 %
Non tobacco	30.0	27.0 %

Sex wise product wise incidence

Sample surveys in 7 selected areas with high prevalence of oral cancer showed

Figure - 34

Habit of chewing in 11 - 55 % in males
10 - 39 % in females

Habit of smoking in 8 - 77 % in males
(not common, 2 - 12 % in females).

Risk ratios of incidence of major tobacco related diseases among smokers in relation to non-smokers, have been given as follows :

Figure - 35

a) Major tobacco related cancers

Site	Risk Ratio Chewer	Smoker	Chewer & Smoker
Oral cavity	6.0	2.8	10.1
Oropharynx	3.3	11.8	31.7
Hypopharynx	6.2	3.6	16.9
Larynx	4.6	7.7	20.1
Oesophagus	2.5	2.2	6.2
Lung	-	14.9(bidi) 10.2(Cig.)	-

(Jussawalla et al. 1971 and 1979)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

b) Cardiovascular diseases (CVD)

Figure - 36

Risk Ratio

	Bidi Smoker		Cigarette Smoker	
	CHD	MI	CHD	MI
Case-control study	3.1	3.8	2.5	3.3
Cohort of blue collar worker	3.3	3.0	3.0	2.8
Cohort of white collar worker	-	-	1.1	1.6

CHD = Coronary heart disease
MI = Myocardiac infarction

(Based on 2 studies, one case-control and another cohort by Notani et al., 1989)

c) Chronic obstructive lung cancer (COLD)

Figure - 37

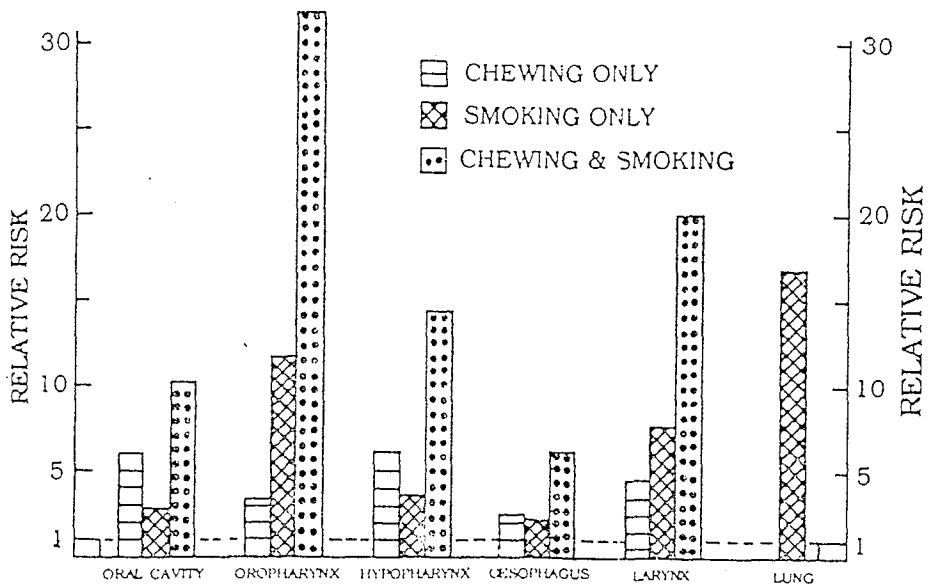
Risk ratio

Cigarette smoker	3.3
Bidi smoker	3.9
Hookah smoker	9.7

(Based on one study in Chandigarh by Malik and Wahi, 1980)

Figure - 38

Relative risk of developing cancers by site and tobacco usage



(Sanghvi, 1989)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Cancer of oral cavity

Distribution of high incidence of oral cavity and cancer of the pharynx, as a % of total cancers by principal countries of prevalence, given as follows : Figure - 39

Type	(a) Hong Kong	(b) Bas-Rhin (France)	(c) Bombay (India)	(d) Bangladesh M	F
Buccal cavity & Pharynx	41.6	40.5	30.3	22.0	16.3
Lip	0.3	0.2	0.3	0.9	0.8
Tongue	2.4	7.4	10.2	4.7	3.1
Salivary gland	1.0	1.0	0.4	1.6	1.3
Mouth	2.7	9.6	5.8	4.1	8.3
Oropharynx	1.0	11.6	4.7	4.1	2.3
Nasopharynx	1.3	10.2	8.2	1.1	0.5

(a), (b) and (c) by Staszewaski (1986), mentions other major prevelance in Latin countries and among Blacks in USA.

(d) by Rahim (1986), Research Programme (CERC) 1976-81.

Notes : Hong Kong, France & India have considerable divergence in tobacco usage. In the first two countries the habit of chewing tobacco is absent. Dietary habits are different in all the 3 countries. Hong Kong & France have heavy alcohol usage. In India, tobacco is used with betel quid. Some alcohol consumption reported.

In B'desh, the general sex ratio in cancer is male to female 3 to 1, whereas in the case of oral cancer, this is 1:2. Chewing and smoking and their composition, similar to neighbouring countries in S.E.Asia. Oral hygiene the same. No Khaini is taken. Alcohol is prohibited. Age distribution of oral cancer for both sexes peak at 45-54.

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Mortality in India

The problems of identifying the cause of death arises from the fact that only a small proportion of the dead population is medically certified :

Figure - 40

Medically certified deaths as % of total registered deaths under Scheme of Medical Certificate of cause of death, 80 - 84.

	All India
1980	13.61
1981	13.66
1982	15.72
1983	14.58
1984	17.08

A 1984 study on All-India distribution of death by age, sex & major causes showed :

Figure - 41

		Male	Female	Total	% of total
1	Infection & parasitic	27,000	17,500	44,500	21.9
2	Neoplasms	5,300	3,200	8,500	4.2
3	Endoctrine, Metabolic diseases & immunity disorder	3,000	2,700	5,700	2.8
4	Diseases of blood & blood forming organs	2,700	2,600	5,300	2.6
5	Mental disorder	103	45	148	-
6	Diseases of the Nervous system & sense organs	4,900	3,300	8,200	4.0
7	Circulatory system	26,839	15,743	42,582	21.0
8	Respiratory system	11,605	8,600	20,205	9.9
9	Digestive system	7,300	2,900	10,200	5.0
10	Genito-urinary	1,900	1,100	3,000	1.5
11	Pregnancy related	-	2,090	2,090	1.0
12	Skin/subcutaneous tissue	208	137	345	-
13	Musculoskelateral & connective tissue	68	56	124	-
14	Congenital disorder	1,012	684	1,696	0.8
15	Prenatal period	9,520	6,800	16,320	8.0
16	Ill defined	9,600	770	10,370	5.1
17	Injury & poisoning	9,300	6,900	16,200	8.0
	Total	1,21,075	82,360	2,30,045	100.0

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

1986 Mortality (some major causes, % to total deaths)

Respiratory	-	7.7
Central nervous system	-	19.7
Circulatory	-	3.7

Percentage distribution of death by sex for different age groups to total deaths by different cause groups - All-India (under Medical Certification Scheme, 1984)

Figure - 42

	Age groups			
	Less than 1	25-44	45-64	65+
Cancer				
Male		12.3	49.8	31.2
Female		17.9	52.1	29.7
Respiratory				
Male	27.8	8.2	21.3	21.7
Female	33.7	8.2	15.8	19.7
Circulatory				
Male			42.5	37.6
Female			32.8	44.3

(blanks above denote insignificance)

Percentage distribution of deaths by sex for different age groups, to total deaths by different cause groups, 1984 as above.

Figure - 43

	Age groups				
	1-4	5-14	25-44	45-64	65+
Cancer					
Male			3.1	8.1	7.1
Female			3.3	16.3	6.9
Respiratory					
Male	15.1	4.7	5.8	7.5	10.4
Female	17.8	15.5	5.2	8.9	10.3
Circulatory					
Male			15.3	34.5	41.3
Female			15.4	33.5	41.1

(blanks above denote insignificance)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

A survey on the cause of rural deaths, 1986 showed the following :

Figure - 44 (Percentage)

Cause	Reported Death No.	Age 15-24	25-34	35-44	45-54	55+
1 Asthma & bronchitis	1,588	1.0	2.4	5.1	11.9	73.7
2 TB of lungs	966	7.4	17.6	20.8	20.7	29.0
3 Pneumonia	965	1.6	2.8	1.7	1.8	6.5
4 Heart attack	886	4.6	7.1	11.2	20.9	54.4
5 Anaemia	557	4.3	5.4	4.7	9.7	34.1
6 Cancer	529	1.9	6.4	14.0	21.9	51.4
7 Gastro Enteritis	397	7.5	6.0	3.3	47.5	22.7
8 Dysentry	356	7.5				
9 Typhoid	334	10.8				
10 Malaria	296	8.8				
All cases	18,262	4.8	5.0	5.9	89.1	46.2

Figure - 45

Causes of death (rural) Annual Report, 1986, Serial 3, No.19

(Percentage)

	82	83	84	85	86
Accident & injuries	5.9	5.5	6.0	6.3	7.0
Natural death	1.0	1.2	1.0	1.2	1.0
Fever	9.3	10.2	10.3	9.3	10.4
Digestive	7.3	7.4	7.4	7.6	7.7
Coughs (respiratory)	19.7	20.2	20.3	20.8	19.7
Central nervous system	3.7	4.5	3.8	4.2	3.7
Disease of circulation	8.9	9.1	9.6	9.9	9.0
Other clear symptom	8.0	7.4	7.8	8.9	8.6
Serility	11.7	11.0	11.0	10.3	10.5
Others	22.6	23.2	22.3	21.5	22.1

Figure - 46

Death by Dysentry & Gastro Enteritis, 1987

	Dysentry		Gastro Enteritis	
	Cases	Deaths	Cases	Deaths
Total	87,41,081	2,109	13,38,594	4,501

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Tobacco related mortality

Figure – 47

a) Estimated tobacco related deaths by cause in the adult (≥ 15 years) population of the country (1986)

Cause of death	Total deaths ('000)			Avoidable deaths ('000)		
	M	F	Total	M	F	Total
Cancer(a)	77	33	110	61	8	69 (63%)+
CHD	290	160	450	97	4	101 (22%)+
Cerebro-vascular	75	75	150	25	2	27 (18%)+
COLD	250	100	350	150	7	157 (45%)+
Total	692	368	1060	333	21	354
			(21%)	(63%)	(21%)	(56%)
All cases			5000	530	99	629
			(100%)	(100%)	(100%)	(100%)

(a) in upper alimentary & respiratory tract

(Notani et al. 1989)

[Sapru, working on Registrar General's report :

(a) Heart diseases 51% of all deaths = 500,000, all ages (1986)
(15+ = 450,000 above)

(b) COLD 26.3% of all deaths = 378,000
(15+ = 350,000)

c) All cancer 2.52% of all deaths = 247,000 (1988)
(Age-sex-specific = 293,000)]

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

b) **Cancer by site (tobacco related)** - Bombay Cancer registry

Figure - 48

Estimates of cancer deaths and avoidable deaths of tobacco related sites in the country (1986 pop.)

Site	Estimated No.of cancer deaths (in '000) Total deaths			Avoidable deaths		
	M	F	Total	M	F	Total
Oral Cavity	11	9	20	9	3	12
Pharynx & larynx	27	7	34	25	2	27
Oeso-phagus	17	13	30	9	2	11
Lung	22	4	26	18	1	19
Total	77	33	110	61 (79%)	8 (24%)	69 (68%)

(Notani et al. 1989)

c) **CVD and COLD** (1986 pop.)

Figure - 49

Cause of death	Range in total deaths ('000)	Range of avoidable deaths ('000)
Ischemic heart disease	300 - 700	66 - 151 (22%)
Cerebrovascular	120 - 200	22 - 35 (18%)
Other diseases of arteries	11 - 22	-
Bronchitis, emphysema, asthma	250 - 460	110 - 210 (45%)

(Notani et al. 1989)

d) COLD

Bombay city data : 400,000
(Notani et al. 1989)

: 250,000 to 460,000
(Mahashur, 1989)

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Role of WHO

Recommendations of the 1974 WHO expert committee on smoking and its effects on health

Governments should accept the responsibility of carrying out smoking control action by their own agencies and of stimulating nongovernmental organisations to take action also. Action should include the dissemination of information, support for activities to help people to stop smoking, the promotion of legislation if further powers are needed, and research.

Governments should develop a procedure for identifying whether or not a serious health problem associated with smoking either exists in the country or may be anticipated, and, if there is such a problem, the government should consider the establishment of a central committee or other appropriate machinery to coordinate and supervise specific programmes for the control and prevention of tobacco smoking.

Objectives should be as follows :

as few young people as possible should start smoking and those doing so should start as late as possible;

as many smokers as possible should be encouraged and assisted to stop smoking;

those who are unable to stop smoking should try to reduce their exposure to such harmful substances in smoke as tar, nicotine, and carbon monoxide;

to require that packets of cigarettes and advertisements carry an effective warning statement that smoking is dangerous to health; provision should be made for the information given and the form of wording used to be varied from time to time so that the message does not become stale;

to determine the magnitude and nature of the problem of smoking, and to assess the smoking behaviour and the attitudes towards smoking of the general public, of the health and education professions, and of other opinion leaders;

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

to determine, if possible, the cost to the country of smoking by calculating the excess of work-days lost by smokers, both from absence due to sickness and from premature death, and the cost of caring for those made ill by smoking, as well as the decrease in morbidity and mortality that could be expected to follow a reduction in smoking. These figures would provide a basis for planning, evaluation, and budgeting of programmes for the control of smoking.

Non-smoking should be regarded as the normal social behaviour and that all action which can promote the development of this attitude be taken.

There should be a total prohibition of all forms of tobacco promotion.

Promotion of the export of tobacco and tobacco products should be discouraged. Tobacco-growing and manufacturing industries should be progressively reduced in size as rapidly as possible,

Governments should recognise the serious dangers for smokers in certain industrial occupations and develop special programmes to eradicate smoking from these industries, introducing legislation where necessary. The synergism observed between smoking and certain occupation implies the need for careful monitoring in industries where toxic inhalation is a problem and indicates the need for research in this area.

Upper limits should be established for appropriate emission products of cigarettes. These limits (currently for tar, nicotine, and carbon monoxide) should be progressively lowered as rapidly as possible. Every tobacco should contain a health warning and information on emission levels.

The developing countries that have a recognisable smoking problem should attempt to control it by the means elaborated in this report, and countries so far without such a problem should give high priority to policies directed at the prevention of smoking.

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

This implies the development of data collection systems to delineate the problem, the adoption of the necessary legislation, and the use of educational techniques suitable to the sociocultural situation, particularly those where communication is difficult.

No country should allow a tobacco-growing or manufacturing industry to be developed. Where such an industry exists, priority should be given to the development of subsitute crops, with international cooperation.

The WHO has anticipated approach of the opposition (against smoking control) as follows :

The approach of the "Opposition" to smoking and health problems is predictable and its arguments may be considered, broadly speaking, to fall into seven main categories :

1) denial of the evidence on smoking and disease;

2) claims that other, more urgent problems should be attended to instead;

3) claims that smoking is a matter of personal choice and smoking control activity is an infringement of essential human freedom;

4) claims that any proposed action would be ineffective;

5) claims that the tobacco industry can be persuaded to act responsibly;

7) claims that tobacco advertising is aimed only at brand-switching and not at increasing sales.

Resolution WHA 39.14 adopted in 1986 by the 39th World Health Assembly mentioned "the use of Tobacco in all its form is incompatible with the attainment of health for all by the year 2000 AD."

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Indian Govt. tobacco control measures

Indian govt. strategies to control tobacco consumption have taken the form of socio-behavioural interventions (banning tobacco in public places etc.), pharmocological and psychological interventions (quality aspects of tobacco products, TV and radio propaganda etc.) economic interventions (gradual price rise of tobacco products etc.) and political actions.

The Cigarette Act of 1975 seeks to check health problems associated with tobacco usage, attempts to regulate the production, supply and distribution of cigarettes besides making it mandatory for manufacturers and retailers to prominently display the statutory warning, "cigarette smoking is injurious to health", on cartons and hoarding.

From 1 Apr 1976 all manufacturers and persons trading in cigarettes are required to display prominently the statutory warning "Cigarette Smoking is injurious to health" on all cartons and packets of cigarettes that are put in sale.

Most state governments in India have promulgated laws prohibiting smoking in closed areas such as cinemas, buses, educational institutions and hospitals etc. Some states engaged primary health workers in the early detection of cancer during their routine house to house check up.

National Cancer Control Boards at the centre, and state Cancer Control Boards at state levels carry out primary prevention of tobacco related diseases.

With effect from 14 Jun 1990, cigarette shops were removed from govt. buildings.

In order to protect the non-smoking public from the hazards of passive smoking, at least in public places where large number of people are present for prolonged periods, it has been decided to prohibit tobacco smoking.

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

To start with, the ban was imposed in a few selected places like hospitals, dispensaries and other health-care establishments, educational institutions, conference rooms, domestic flights, air-conditined sleeper coaches in trains, subsurban trains and air-conditioned buses.

A more restrictive and punitive anti-tobacco legislation is being comtemplated. The proposed legislation includes a rise in the price of tobacco, banning of tobacco advertisement in the media and public places, making it unlawful to sell tobacco products to those below the age of 18 years, sale of cigarettes in packets as opposed to selling them singly and prohibition of the sale of tobacco in and around educational, health and religious institutions.

Besides, it is proposed that tobacco acreages will be reduced significantly and to totally ban advertisements (including sponsor-ships).

The ICMR has claimed that health care cost in 1986-87 on account of tobacco related diseases exceeded govt.'s excise revenue by Rs.685 crores and amounted to Rs.2419 crores.

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

Discussion

We begin with an examination of what have been claimed as tobacco-related diseases.

Cancer will be discussed in two parts: lung cancer with which cigarette has been associated in the developed countries (bidi and cheroot also to be associated with this disease in India) and oral and pharyngeal cancer which is the main cancer-type among male in India associated with the consumption of bidi and chewing tobacco. These will be followed by the two other main diseases associated with tobacco, viz., cardio-vascular diseases (CVD), including coronary heart diseases (CHD) (circulatory diseases) and chronic obstructive lung diseases (COLD) (respiratory diseases).

Lung cancer

Following early studies (pages 1 & 2) on smoking and lung cancer, the US Surgeon General conclude in 1964 that cigarette smoking was the major cause of lung cancer in men, with the Royal College of Physicians reporting (page 8) on Smoking and Health (1971) the highest mortality ratio for this disease. However, Fisher and Burch (page 11) and Resenblatt (page 12) disagree with the Surgeon General's conclusions. The Royal College of Physicians admit that only a minority of heavy smokers get lung cancer. Buhler (page 12) does not accept smoking - causation of lung cancer. Fisher (page 12 & 13) shows that its increase in white females was the same for smokers and non-smokers. The Bethesda (1985 study) (page 13) fails to develop squamous cell lung cancer over a 9-year old study on 10,000 mice. Furst (page 13) corroborated the Bethesda findings in his animal studies. Eysenck criticises (page 15 to 22) the methodology of arriving at risk ratios and a few criteria for causal significance of tobacco with concerned diseases. He urges examination of other especially genetic factors, before coming at conclusions.

The WHO (page 26) state that laboratory models for the production of diseases due to smoking are not satisfactory other than for lung cancer. WHO (page 43) state 'in one study involving long term exposure of rats to cigarette smoke, tumours of the respiratory tract were induced',

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

'Studies in rabbit and dogs of whole cigarette smoke were inadequate for evaluation', 'Experiment on carcinogenically of gaseous phase of cigarette in hamsters and rats resulted in negative or inadequate findings. An increased incidence of lung tumours was observed in one study in mice', thus proving studies for lung cancers on animals have been extremely few in number. Also the type of tumours produced is not mentioned. Yet WHO state (page 42) 'there was sufficient evidence that inhalation of tobacco smoke condensate cause cancer in experimental animals. Now there is sufficient evidence that tobacco smoke was carcinogenic to human. The occurrence of malignant tumours of the respiratory tract and of the upper digestive tract is causally related to the smoking of different forms of tobacco (cigarette, cigar, pipe, bidis), the WHO raise the questions of genotypes who develop lung cancer.

The tar yield (1982 figures) of Indian cigarettes (page 28) at 17-22mg/ cigarette, although on the high side in comparison with similar yields in most developed countries, was not inferior to France and Italy.

WHO (pages 29-30) conclude that lung cancer has "association reflecting causation" and point out (pages 32-33) that variations in smoking habits, dose of consumption, and duration of smoking are important variables.

Dose-inputs of any form of tobacco per adult capita is among the lowest in the world and on this logic the incidence of, lung cancer should be very modest. WHO point out (page 32) that until smoking has been common for many year the incidence of the diseases may not appear at first sight.

Bidi smoking in this country in India has been prevalent for over 100 years and cigarette, around 60 years and no major incidence of lung cancer has yet appeared across the country attracting attention.

The comparatively low incidence of lung cancer deaths among cancer deaths in Japan in comparison with USA, West Germany, France and U.K. (pages 34-38) raises a very important question of the morbidity's causal relationship with cigarette smoking. At a per capita consumption of cigarette per adult population of 3210 per annum, death in Japan by lung cancer of all cancer deaths was 15.4% This was 26.2% in USA at

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Source: https://www.industrydocuments.ucsf.edu/docs/kqhl0000

an incidence level of 3250 and 16% in W. Germany, at an incidence level of only 2320, 28% lower than Japan.

Lung cancer as a percentage of new cancer is 9.4% in developing countries as against 15.8% in the developed countries, presumably reflecting among other factors, a lower incidence of smoking. WHO have reported (page 41) that the risk of lung cancer is particularly dependent on duration of smoking. The longer the time period during which a major proportion of adult in a population have smoked the greater is the incidence and mortality from the disease in the population, risk being proportional to the number of cigarette smoked.

WHO also mentions (page 42) of lung cancer risk increasing with high dose of exposure to alcohol, suggestion for the control of which in terms of lung cancer control either from WHO or Indian Govt. has been notably absent.

Incidence of all smoking (pages 52 & 53) among rural male in India is 24% and urban male 18%, with rural and urban female smoking, negligible. Age-group wise the incidence of smoking amongst rural male is 18% in the 15-24 age range, 52% in the 25-44 range and 56% in the 45-59 age group. In the urban centres it is 9%, 39%, 45% and 33% respectively for the age groups; overall inicidence, of all ages being 25% among rural male and 20%, urban male.

Teen-age consumption in India is less than in most developed countries. Hence, as a primary target audience for smoking control, they have a low priority. Further, assuming that tobacco-related diseases need an exposure period of around 20 years, diseases should arrive progressively more at say 45 plus age groups, at least upto 65 years. However, figures 42 and 43 (page 68) show a decline in cancer after the 45-65 age group. Site wise, age wise figures are not available. There is some confirmation of our argument in figure 44. However, without sex or site-wise classification, no conclusion could be drawn.

The Indian incidence of consumption of cigarette (pages 57,68 and 59) has been declining since 1950-1955. Total tobacco consumption is down. Within the group since 1970-75, bidi consumption has gone up. For

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all others, there has been a decline since 1965-70. Hence the dose inputs of tobacco into the Indian population have been declining. Page 58 shows that among the listed 37 countries, the average consumption of cigarette per adult population is the lowest, including bidi and chewing tobacco after adjusting for quantity in Kg. and unit sale, at 600, the computed incidence of Indian consumption is still among the 7 lowest consuming countries.

The incidence of lung cancer in India varies between 6 to 8% of all new cancer cases (figures 31 and 32), a much lower level than most developed countries and the average for the developing countries.

The WHO estimate (page 39) that globally there are 143 cases per 100,000 population. Applying this ratio crudely to the current Indian population of 840 million the total cancer cases in India should be around 1.2 million. At an incidence rate of 8% of lung cancer of all cancer cases, the number of such cases in India should not exceed 96,000 persons.

Due to non-availibility of data (only 17% mortality being medically certified, figure 40), mortality by cause figures are difficult to obtain in India.

A few studies under medical certification have been made. Figure 41 (page 67) shows death by neoplasms is 4.2% of all deaths in India. According to figure 44 (page 69), death by cancer is 2.9% of all rural deaths.

In conclusion, although the US Surgeon General says that smoking is the major cause of lung cancer in men and the WHO has placed under its category A, suggesting 'association relecting causation' it does not necessarily follow that this should be necessarily so in Indian condition.

The evidence of occurence of lung cancer in Japan (page 35) casts significant doubts in the causation theory being accepted a level of dosage of tobacco automatically causing cancer. The needle here points to genetic, dietary or other unknown factors. In any case, the per adult capita Indian consumption at 160 cigarettes or an equivalent of 600 cigarettes, both among the lowest in the world, should not qualify

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cigarette (and partly bidi) as a serious hazard for lung cancer. With the consumption stagnant or declining, this is unlikely to be so in the forseeable future.

Oral Cancer

The US Surgeon General concludes (1964) (page 4) that cigarette smoking is the cause of cancer oflaryngeal and oral cancer. The Royal College of Physicians (1971) arrive at mortality ratio of 5.4, 4.1 and 3.4 for cancers of the larynx, oral cavity and the oesophagus (page 8).

The WHO (pages 29-30) put cancer of oesophagus, lip, tongue, mouth, pharynx anf larynx under category C, denoting 'positive association (of tobacco) of uncertain character'. Yet, they mention (page 41) that 'tobacco smoking is an important cause of oral, oesophageal, hypopharyngeal, laryngeal and oesphhageal cancers'.

In the developed countries the incidence of this combined group of all cancers is 10.5%, as against 25.2% in developing countries (page 40). WHO says that oral cancer is South East Asia is a result of chewing tobacco and betel nuts (page 41). It also introduces the concept that the rise of these cancers is substantially increased in conjunction with high dose of exposure to alcohol.

In hamsters, various experiments demonstrated reproducibly the induction of laryngeal carcinomas (WHO) (page 43). In India, Niblock (1902) and Orr (1933) report incidence of oral cancer among betel leaf chewers (page 46). Sanghvi et al. (1955) find a significant role of bidi and chewing tobacco in incidences of oral cancer, with the incidence being higher where both bidi and chewing tobacco were consumed (page 46). Notani et al. (1989) say that 63% of morbidities of cancer in the upper alimentary and respiratory tracts are tobacco related. 'Since they form only 1% of the total morbidity, the overall impact is limited' (page 48).

The dose - impact of chewing tobacco in the target audience is small, because only 8% or rural males, 5% of urban males, 5% of rural females and 3 % of urban females chew tobacco (page 50).

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As in smoking, chewing habit in India starts late at 25-44 age-level and peaks at 45-59 age-level (page 54).

At a per capita monthly expenditure of only 0.02 Kg. in rural areas and 0.01 Kg. in urban areas, the dose of chewing tobacco among the population is extremely modest. However, the expenditure per month on bidi @ 50.58 units in rural areas and 40.29 units in urban areas is comparatively higher, although modest in terms of smoking consumption in developed countries at around 17 bidis in rural areas and 13 bidis a day (page 55).

Consumption of chewng tobacco has been declining since 1955-60 (page 62).

Sanghvi (1959) arrives at an estimate of total mouth, pharynx, larynx and oesophagus cancer population in 1986 of 142,000 (page 62, figure 30), but does not explain how he arrived at the crude incidence rates.

The Tata Memorial Hospital (190-72) mention that among males, the incidence of oral, pharynx and larynx cancer is 62.5% and 23.3% among females, of the cancer cases (page 62).

The weighted average from the National Cancer Registry in Bangalore, Bombay, Chandigarh, Dibrugarh, Madras & Trivandrum. for 1986 for oral cavity, pharynx, larynx and oesophagus is 45% for males and 18.9% for females, of all cancer cases (page 63).

The risk ratio of cancer in these types is the highest when the consumer is both a chewer and smoker.

Figure 39 (page 66) is self explanatory, suggesting that oral cancer occurs only as an inter-play of various factors (Staszewaski, 1986) and not due to tobacco consumption alone. The part played by tobaccoin oral cancer needs further investigation.

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Cardiovascular disease (CVD)/Coronary heart disease (CHD)

The 1964 Surgeon General (1967) finds that smoking would cause death from coronary heart disease (page 2). He finds (page 4) that smoking increased mortality from CVD (stroke) and atherosclerotic peripheral vascular disease.

Marks and Emerson (1974); Pollock, Swiss study (1951-76); Handley and Teather (1974); Sterling (1977) claim no adverse effect of tobacco on CHD (page 13) with Schievelbein (1973) agreeing after experiments on animals. Keys (1962) also agrees (page 21) with their views.

Seltzer (1968) concludes that duration of cigarette smoking had no correlationship with CHD in terms of evidence upto 1984 (page 22). Commenting on the Framingham data, he concludes (page 24) that the risk of developing CHD increases with the total duration of smoking, this important study having concluded that cigarette smoking is only acutely (not chronically) connected with cigarette smoking, suggesting that it had no deleterious effect on those with a normal, healthy coronary apparatus (page 22).

Eysenck (page 25) claims that personality factors were more predictive of death from cancer or heart disease than was smoking, and in particular, ways of coping with stress (page 25).

In an important differentiation, WHO (page 29) splits categories, putting ischaemic heart disease, respiratory heart disease, aortic aneurysm and peripheral vascular disease in Category A where association with tobacco reflected causation and myocardial degeneration, hypertension, arteriosclerosis, cerebral thrombosis and other cerebrovascular diseases in Category C where association with tobacco was positive but of an uncertain character.

S.P.Gupta (1989) states (page 47) that reliable data on the effect of tobacco on cardiovascular diseases was unavailable and consequently, research data had to remain tentative.

Notani et al. (1989) estimate (page 61) mortality of CVD in the 40-49 age group at 320,000 to 448,000 cases and CHD at 1,300,000 cases

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correcting the latter to 1,270,000, being 21% of the total number of tobacco-related cases. However, the estimation was based on two studies in Haryana, one rural and the other urban. Most data on CVD/CHD originate from Bombay Municipal Corporation and Registrar General's records, both inadequate for projection into all-India estimations. All-India 1984 diseases of the circulatory system (Figure 41) were 21.0% of all deaths, second only to infections and parasitic diseases. Sapru (figure 47, page 70) says 51% of all deaths was on account of CHD. Notani et al. 1989 estimated avoidable CHD/CVD mortality at 22% of total avoidable deaths.

The all-India percentage distribution of death by sex for different age groups to total death by cause shows, under Medical Certification Scheme (1984) (figure 42), a decline in deaths for males and increase in females from the 45-64 to 65+ age groups, which is contrary to what should happen if such deaths were entirely tobacco related, with consumption progressively increasing with age from 15 to 59 years (figure 23) for both males and females.

The prevalence of CVD and its link to tobacco & types of tobacco in India has been difficult to estimate (Notani, 1989). Risk factors as suggested by the U.S. Department of Health and Human Services (1983) linking smoking with hypertension and elevated cholesterol levels, acting synergistically and thus to cardiovascular diseases have been assumed in many Indian studies. CHD/MI mortality risk ratios are not available in India. Incidence risk ratios and mortality risk ratios are also not available for cerebrovascular and perepheral vascular diseases (Notani 1989). Risk factors have to established first locally, at a particular time. Eysenck has pointed out (page 17) that there was a r = 0.75 correlation between ischaemic heart diseases and milk consumption and also a correlation of 0.75 between consumption of refined sugar and ischaemic heart diseases, over 45 countries. Therefore, the tobacco related risk has to be adjusted after eliminating other risks.

Stray Indian researches, working on small samples (Bansal et al. 1970, Sharma et al. 1976, Chiniah et al. 1979, Dewan et al. 1974, Jayant et al. 1983, Bordia et al. 1979) have been extrapolated often on a crude or only 15+ basis into national estimates of the disease. Similar important inferences have been made regarding tobaco product type.

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Diognostic data specially in rural areas would often be faulty and medical certification of death assigning cause is largely absent.

We therefore conclude that tobacco relation to CVD/CHD in India should be treated as tentative, not requiring control on tobacco or types of tobacco.

Chronic obstructive lung disease (COLD)

In his 1964 report the US Surgeon General suggests that cigarette smoking causes chronic bronchitis and in his 1967 report, 'it was the most important cause of chronic non-neoplastic bronchopulmonary disease in the USA (page 2)'.

The Royal College of Physicians conclude (page 8) in their 1971 report that there was a 6.1 mortality ratio for ronchitis and mphysema due to cigarette smoking (page 8).

Seltzer (1975) finds that there was a difference in mean value of the pulmonary function among White nonsmokers and smokers, although it was the same among Blacks and Orientals (page 21).

WHO (1986) state that it is not possible to show whether the particulate or the vapour phase is more important in the production of (vascular and) chronic obstructive lung diseases (page 26). However, WHO have put COLD in Category A, signifying association reflecting causation (page 29).

WHO point out (para 2, page 32) that 'it is however uncertain how far the effect of smoke in producing COLD is modified by background levels of atmosphere pollution.'

WHO (1986) point out that the risk of disease and the duration of smoking has been worked out most clearly (cancer of the lung) and COLD (pages 32-33). On this logic, COLD should affect mainly the 45+ population who have had about 20 years of exposure. In India (page 68), however, Indian mortality was 27.8% (male) & 33.7% (female)for children of the age group of less than 1 year) (figure 42) and in pre-45 age group (figure 43) it was 25.6% for male and 38.5%, for female.

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Jindal and Malik (1989) find a relationship between cheroot smokers in Shimla hills and South-East Orissa and COLD (page 47).

Studies by Charan in North India, Punjab (1966), Thiruvengadam et al. in Madras city (1979), Viswanathan & Singh in urban and rural Delhi (1977) report wide variations in the disease. Studies in North India, specially in Chandigarh, by Malik (1978 & 1982) and Joshi et al. (1975) bring out differences (and anomalies) in the prevalence of the disease in rural and urban areas.

Sapru (1983) point out that there was a difference in the disease pattern beteen South and North India. Jayant's 1989 estimate of prevalence and mortality due to COLD in India, is based on 15+ population which should be corrected to 45+, different distribution being given in Fig. 18 (page 49).

Notani (1989) mentions that the estimated 400,000 COLD deaths are extrapolation of age-sex-specific death rates of Bombay city to the whole population. In view of Joshi and Sapru's foregoing reservations that differences exist in rural and urban areas, North and South India, this has to be treated with reservation. Also his estimate (1989) that 45% of COLD deaths originate from tobacco usage (page 48) is based on 15+ population, which according to us, should be corrected to 45+ population.

As mentioned earlier the per capita incidence of tobacco in India is low (pages 58-60) because of which, as in the case of lung/oral cancers and CVD/CHD, tobacco's contribution also in the production of COLD cannot be significant.

Figure 44 (page 69) shows no abnormal incidence of death in rural India of Asthma and Bronchitis until the 55+ age group. At the latter level, under Indian conditions, the mortality could easily be due to 'old age', rather than tobacco. If it were tobacco, the 45-54 would have been the first level to be affected.

Figure 45 (page 69) shows Coughs (respiratory) to be steady around 20.1%. Whether it is tobacco related or not the disease remains contained.

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In view of data as above, causal relationship of tobacco with COLD can not be concluded as yet. Neither, any particular tobacco type be identified in production of the disease.

Indian medical researches

Indian medical researches have usually relied on findings in the developed countries as being axiomatically applicable to India. Also, often, risk ratios are assigned without proving causal relationships and projections of all-India figures are made with inadequate samples.

External variables in the studies of the developed countries are different from India and important, their bulk is based on cigarette which in India has a comparatively smaller share in consumption than the indigenous tobacco products.

Sanghvi (1986) calculates relative risks of developing cancer of the upper alimentary and respiratory tracts among the chewing and smoking population, contravening the very logic of an epidemiological enquiry, taking for granted what is to be proved. The risk ratio only provides an evaluation of the importance of the factor in the production of a disease once its causal effect has been proved. It cannot be used by itself to prove the causal relationship, which may differ according to the population in question, time, genetic and other factors.

Major studies by P.C.Gupta et al. (1980 & 1984) assessing excess mortality caused by tobacco usage in India are on a purposive sample, on a population which reportedly had a high tobacco usage. Prevalence of tobacco habit was found to be (62% to 82%) among men and 15% to 65% among women. In the light of NSS 43rd round 1987 (pages 50 to 54) figures, the all - India average incidence of tobacco consumption is found to be substantially lower. Accordingly, his assessments of all-India morbidity should be revised downwards.

No data on cause of death was obtained. Therefore, whether the smoker died from any of the claimed tobacco related diseases. is not known, neither, was it known how many nonsmokers died out of any claimed tobacco related diseases.

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While estimating the number of deaths in their paper on COLD.In India Morbidity amd Mortality Due to Smoking (1989), Jayant and Mahashur's population base is 15+ which should have been 45+. Notani et al. (1989) also assess on the basis of 15+ population.

Sanghvi (1989) uses (page 63) population-based Registries to project country-wide figure. Such registries are biased in that only cancer patients come to such hospitals and do not represent the cancer universe.

WHO

The WHO claim association reflecting causation for 5 claimed tobacco related diseases. However, figures in pages 34-38 on country-wise comparisons of the principal tobacco related diseases indicate that causal factor in lung cancer is still to be proved.

It is a fair question if the WHO is pushing India into wrong priorities, conflicting with more urgent problems of infant mortality, parasitic diseases etc., with an Western disease model based wholly on cigarettes. They ignore tobacco types, usage habits, actual incidence of disease and resource, employment and income generation problems of an under-developed country, and media problems in reaching the target audience.

Indian tobacco control

Decline in the per capita incidence of tobacco consumption started following market forces long before the Cigarette Act of 1975 was enacted (page 57).

However, due to the intruments used by the Govt. borrowed from the developed countries for control of tobacco (and selective use of excise) shifts within tobacco types have occurred.

Controls have in effect, been directed at cigarette although it may not have been necessarily the target audience. Among the tobacco product types, it has the lowest tar and nicotine delivery and lung cancer in India with which principally cigarette is associated (bidi's role in

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this disease not backed by extensive resaerch), is only 8% of all cancers.

At the same time, most of the control intruments have by-passed the problems of oral cancer whose incidence is predominant among all cancers in men with which bidi and chewing tobacco usage has been linked.

Hirayama (1986) suggests nutritional interventions for this disease and Staszwaski (1986) mentions that oral cancer occurs out of an interplay between alcohol drinking, tobacco smoking and chewing, chewing betel nuts, nutritional deficiency and oral hygiene. The Govt. have not initiated any measure on nutritional aspects or alcohol.

Ignoring estimates which as we have shown, have built-in estimation errors, there is no data on the ground on an all-India scale that Cardiovascular/Coronary heart diseases and chronic obstructive lung diseases are as extensive as claimed by Notani et al. (or that they are necessarily tobacco originated). If they were they would have found a place in the National Health Policy 1983 : Health For All in 2000 AD. Targets for prevention of prenatal, infanct, preschool and maternity mortalities, TB, leprosy, blindness etc. have been laid down, but none of the tobacco-related diseases find a place in such targets. Under the circumstances, investments in tobacco control divert resources in the areas given priority in the National Health Policy.

Govt.'s investment in tobacco control would have been on s sounder footing if surveys were conducted first on the tobacco related disease problems. WHO recommends that it be found out by the Govts. if such diseases exist or are likely to exist. The Indian Govt. does nor appear to have followed this advice. Second, controls should have preceded a study on tobacco consumers: why they consume it, time of starting, volume of consumption, number of years consumed etc. For the first time, NSS figures have given some all-India statistics which should lead to substantial corrections of the previous estimates. Only such studies would allow for some understanding of causal effects, allow for appropriate decisions disease-wise, urban - rural-wise, income group-wise, age-wise, sex-wise problems and instruments to be selectively used.

It is important for the Govt. to understand why people consume tobacco. Control does not necessarily mean coercion. People must be provided

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with better, acceptable subsitutes to tobacco specially those in remote rural areas. Without such substitutes, there could be a vacuum, tension, stress or tobacco eventually replaced by products like alcohol or drugs.

It has been suggested that tobacco acreages be reduced and released for other crops. The bulk of tobacco is grown in rainfed areas. It produces a better and more stable cash income than cotton, groundnut etc. which could technically replace tobacco. It does not compete with food crop.

Inter-crop shifts are presently mediated through market forces and if the Govt. wishes to deliberately reduce tobacco acreage, farmers will need to be convinced. If there is to be coercion, the problem will be, how to bring about the change, keeping legal provisions in mind.

A solution might be to restrict the demand for the high tar, high nicotine products through differential taxation imposed at the grower level and thus to restrict demand through pricing.

Further restrictions or banning of tobacco advertisement will prevent consumers from being informed of better quality products. In this connection we refer to the growth in cigarette consumption in Eastern Europe, USSR and China (figure 27, page 59). These command economies, run on the principles of social justice and maximum health care have allowed steady increases in consumption. There has been no advertisement of cigarettes in these countries.

An important unintended effect through neglect of the anti-tobacco posture of the Govt. has been on exports. Between 1975 and 1988, growth in Indian exports was only 21%, as against global 35%, Italy's 809%, Greece's 173%, Malawai's 270%, Zimbabwe's 243%, Brazil's 224%. If India could have at least maintained the average export level of 112 m. Kg. of 1981 and 1982, the value would have been around Rs.670 crores at current prices.

Luthra et al. (1990) mention a figure of Rs.2418.82 crores being spent on diagnosis and health care for tobacco related diseases, but have given no break-up of the diseases covered, allocations among them,

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capital-revenue expenditure break-up, and the morbidity figures that have been estimated.

If they be based largely on cancer, 22 specialised hospitals in 14 States have a patient load in 1987 of 46,524. On a per capita basis Rs.2418.82 crores works out to Rs.5,19,946.

An important cancer hospital which we investigated mentioned an annual recurring expenditure not exceeding Rs.1.50 crore, with capital expenditure around Rs.2 crores, but varying with new instruments being introduced. The hospital has a patient load of 7000 per annum indoor and outdoor, 210 beds utilised through the year and 40 specialists. Per person basis, the revenue to expenditure allowing for depreciation on instruments, comes to Rs.17,000/- per annum.

The total per capita private consumption expenditure in India on medical care and health costs in current prices was Rs.5650 crores in 1986/87. It is difficult to estimate cause-wise morbidity as existing in India. Taking mortality figures given in Figure 41 (page 67), on the assumption all such mortalities are wholly tobacco-related which they are obviously not, deaths by cancer, circulatory and respiratory diseases are about 35% of death by all causes. Thus, the proportionate expenditure should be Rs.1977.5 crores.

The Govt. of which the Health Ministry is a part has the dual responsibility of improving health and preventing hunger.

There is so far no hard data in India as yet that the current incidence of tobacco related diseases is beyond acceptable limits. Without such an evidence, further imposition of punitive control measures on tobacco growing, primary processing, manufacturing, distribution and sale, will result in social and economic costs exceeding health gains.

Even the current stagnation has seeds of the industry turning sick because of inflation. Indeed there is a risk of some of the sectors moving away from tobacco before the Govt. will want them to taking into view larger macro-economic considerations like employment, excise, exports etc.

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If tobacco is to be phased out, alternative arrangements will have to be made at the same time, for those in various sectors like, growers, manufacturers and consumers. Inter-sector balances have to be maintained.

This will be difficult to achieve in India's developing economy perspective. Alternative employment and income opportunities are few and there are severe resource contsraints. The political economy in which the tobacco system operates with many conflicting interests even within its own ministries make for decision-making difficult.

While the current approach to controlling tobacco consumption may or may not mitigate health problems, it has every potential to create hunger all around without such integrated plans.

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Executive Summary

The Royal College of Physicians (1862) and U.S. Surgeon General (1964) were the two important bodies who pointed out that cigarette caused premature deaths and disabling diseases.

These studies have however been subject to criticisms in various researches.

The 1985 study at Bethesda on 10,000 mice failed to reproduce squamous cell lung cancers.

Hans J Eysenck criticised the methodology adopted in the Royal College and similar studies, pointing out that they were causal interpretations of the statistical association between tobacco and tobacco related diseases and that criteria for causal significances of association were not substantiated. He suggested constitutional and genetic factors for explaining the tobacco related diseases.

The Framingham study suggested that tobacco was only acutely connected, not chronically connected with coronary heart diseases, there being no deleterious effect of tobacco on them with a normal, healthy coronary apparatus. Seltzer, commenting on the results of the Framingham study mentioned the strong possibility of a selection bias in the US Surgeon General's 1983 study on CHD.

According to WHO there are about 43 carconogenic elements in tobacco smoke.

The WHO have placed cancer of the lung; ischaemic and respiratory heart disease; aortic aneurysm, peripheral vascular disease and chronic obstructive lung disease in its A Category of 'association reflecting causation'. Oral cancer, which is the dominant cancer site among men in India, has been put in its C Category, having 'positive association of uncertain character'.

The low incidence of lung cancer amongst Japanese men on a comparative consumption level with the JSA, questions the tobacco-causal theory of lung cancer.

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According to WHO, carcinogenicity tests in animals have technical problems. In one study, tumours of the respiratory tract of rats were induced. Other studies were inadequate for evaluation.

Sanghvi et al. showed a significant role of bidis in oral cancer and Bhide et al. gave evidence of several carcinogens in N Rustica.

Incidence of tobacco consumption in India per adult capita in any form is among the lowest in the world and has been declining since 1935. In rural India 34% men use tobacco in some form regularly. This in urban India is 24%. With women consumption is low in both cases.

Hongkong, Bas-rhin (France), India and Bangladesh are among countries which have a high incidence of Oral Cancer. Since the habit of chewing in the first two countries is absent, dietary habits are different in Hongkong, France and India/Bangladesh the tobacco-causal relationship of Oral Cancer has to be questioned.

Age-wise mortality in India does not justify the inference that cancer, respiratory diseases, and circulatory diseases have a significant tobacco-causal relationship.

Tobacco related diseases are dose-specific and usually an exposure of 20 years or so is necessary before morbidity starts. It increases progressively with age.

In India for both cancer and circulatory diseases there is a decline in morbidity after 64 years. In respiratory diseases, its incidence is dominantly in the pre 45 age group.

In India, for lung cancer, genetic, dietary or other unknown factors along with tobacco should be examined for causal relationship. On account of extremely poor incidence of cigarette smokers among adult population, and with stagnating and declining consumption, cigarette does not qualify as a serious health hazard for lung cancer in India.

For oral cancer, alcohol, malnitrution, interplaying with bidi and chewing tobacco should be the setting for examination of incidence of the disease.

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In order to draw conclusions for cardiovascular/coronary heart diseases, and chronic obstructive lung diseases, Indian researches are inadequate. In any case, estimates of the disease suffer from large estimation errors.

Indian medical researches have usually relied on findings in developed countries as being axiomatically applicable to India, and risk ratios have been assigned without prior proving of cusal realtions as obtaining in India.

The Indian measures for controlling tobacco usage have resulted, by implication, in mainly cigarette being affected adversely. They by-pass bidi and chewing tobacco which are usually associated with oral cancer.

Through neglect, exports of tobacco from India have not grown in line with global trends or principal competing countries.

In order that correct instruments could be used to control what have been claimed to be tobacco related diseases, the goverment should conduct surveys on diseases, consumption pattern and, why people consume tobacco.

The government has the responsibility both for improving health and preventing of hunger. The current stagnation in the industry is likely to turn the industry sick because of secular inflation. If tobacco is to be phased out alternative arrangements for employment, income etc. will have to be made at the same time, keeping inter-sectoral balance (backward and forward linkages). The current trend has the potential to create hunger all-around.

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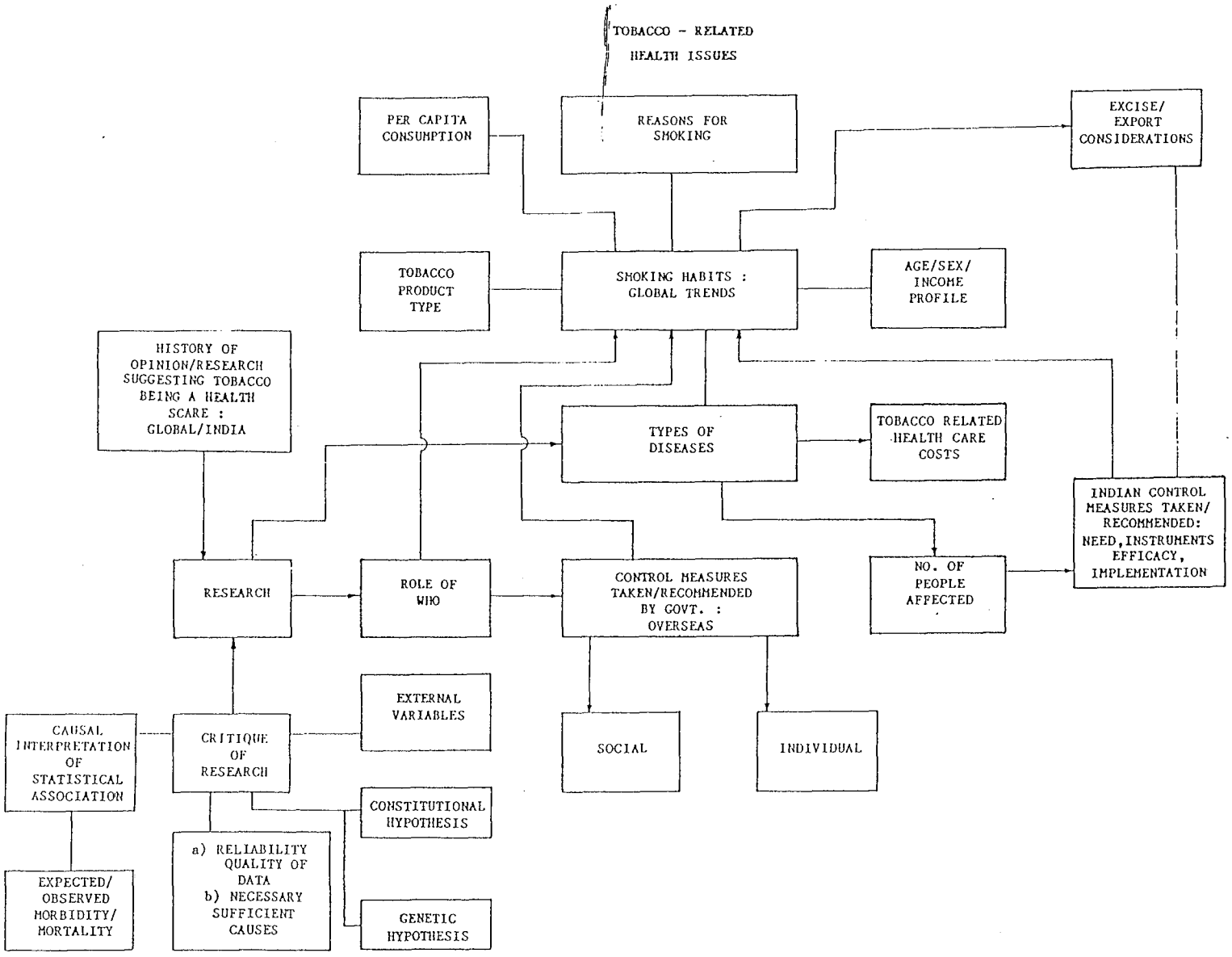

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